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Ohio Cardiovascular and Diabetes Health Collaborative

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Special populations: Patients with chronic mental illness

Thursday, November 19, 2020

Disclosure Statements

- The following planners, speakers, moderators, and/or panelists of the CME activity have financial relationships with commercial interests to disclose:
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  - These financial relationships are outside the presented work.
- All other planners, speakers, moderators, and/or panelists of the CME activity have no financial relationships with commercial interests to disclose.

Special populations: People with chronic mental illness (CMI)

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Objectives

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1) Describe the epidemiology and outcomes of diabetes among people with chronic mental illness (CMI).

2) Describe the impact of treatments for people with CMI, including atypical antipsychotic medications, on control and outcomes of diabetes.

3) List and describe a minimum of 3 strategies to improve control of diabetes among people with CMI

Medical comorbidity among individuals with chronic mental illness (CMI)

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- CMIs such as recurrent depression, bipolar disorder and schizophrenia generally complicate general health outcomes
- CMI is often accompanied by additional mental health comorbidities such as substance abuse and PTSD
- CMI inflates costs
- CMI life-span reduced by 10-30 years.
- CMI have a 1.2 to 4.9 increase in mortality compared to age and sex-matched individuals from the general population resulting from DM, cardiovascular disease, and stroke.

Colton CW, Manderscheid RW. Preventing Chronic Disease 3: A42: 2006

Whiteman, K. L et al. *Psychiatric Services* *67*(11), 1213–1225.2016

Walker, E. R. et al *JAMA psychiatry*, *72*(4), 334–341. 2015

High-prevalence medical burden in CMI

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Modifiable Risk Factors	Estimated Prevalence and Relative Risk (RR)	
	Schizophrenia	Bipolar Disorder
Obesity[1-5]	45–55%, 1.5–2 × RR	21–49%, 1–2 × RR
Smoking[4-8]	50–80%, 2–3 × RR	54–68%, 2–3 × RR
Diabetes[2, 8-11]	10–15%, 2 × RR	8–17%, 2 × RR
Hypertension[2-4, 7-9, 11]	19–58%, 2–3 × RR	35–39%, 2 × RR
Dyslipidemia[2, 4, 11-13]	25%, ≤ 5 × RR	23%, ≤ 5 × RR

1. Allison D, et al. *J Clin Psychiatry.* 1999;60(4):215-220;
2. Fagiolini A, et al. *Bipolar Disord.* 2005;7(5):424-430;
3. McElroy S, et al. *J Clin Psychiatry.* 2002;63(3):207-213;
4. Hennekens C, et al. *Am Heart J.* 2005;150(6):1115-1121;
5. Davidson S, et al. *Aust N Z J Psychiatry.* 2001;35(2):196-202;
6. Ucok A, et al. *Psychiatry Clin Neurosci.* 2004;58(4):434-437;
7. Herran A, et al. *Schizophr Res.* 2000;41(2):373-381;
8. Goff D, et al. *Schizophr Res.* 2005;80(1):45-53;
9. Dixon L, et al. *J Nerv Ment Dis.* 1999;87(8):496-502;
10. Cassidy F, et al. *Am J Psychiatry.* 1999;156(9):1417-1420;
11. Kilbourne A. *Bipolar Disord.* 2004;6(5):368-373;
12. Allebeck P. *Schizophr Bull.* 1989;15(1):81-89;
13. Koro C, et al. *Arch Gen Psychiatry.* 2002;59(11):1021-1026

Diabetes in those with CMI

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- Prevalence of DM in people with schizophrenia, bipolar disorder or schizoaffective disorder is 2-3 fold higher vs. the general population
- Risk of DM in those with depression or depressive symptoms is 1.2-2.6x higher vs without depression
- Age of onset of DM in those with a CMI is 10-20 years earlier vs. the general population
- An increase in well-established DM risk factors in CMI patients partially accounts for much of the increased risk. However, additional factors (disease, treatment) are important as well.

Barnett AH, et al, J Psychopharmacol. 2007

Carnethon MR, et al, Am J Epidemiol. 2003

De Hert M, et al, Eur Psychiatry. 2009

McEvoy JP, et al Schizophr Res. 80(1):19-32.2005

Whiteman, K. L et al. *Psychiatric Services* *67*(11), 1213–1225.2016

Factors which elevate DM risk in people with schizophrenia

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Genetic susceptibility:

higher occurrence of DM in family members of people with schizophrenia

abnormal glucose metabolism

common mechanism proposed for cognitive deficit and glucose metabolism

Neuroendocrine pathways:

hypothalamic axis dysregulation and elevated cortisol in schizophrenia

nutritional deficiencies proposed as common pathway for both diseases

Antipsychotic medications:

effect on hypothalamic regulation, dopaminergic, serotonergic, and histaminergic receptors

other proposed mechanisms: action on pancreatic muscarinic receptor and leptin resistance

Environmental/additional comorbidity:

diet and lack of access to quality foods

inadequate physical activity due to symptoms and social isolation

Anamalie. Int J Endocrinology. Volume 2015 |Article ID 969182 | https://doi.org/10.1155/2015/969182

Assessing medical risk in CMI should include bio-behavioral variables

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- Composite, CVD /general medical burden measures such as the Framingham Risk Score may not optimally assess risk in CMI.
- One review found Framingham not associated with any changes within multicomponent intervention models in adults with CMI. May relate to the fact that the Framingham Heart Study risk scores were determined with a sample that excluded adults with CMI.
- Risk prediction models that include **bio-behavioral variables such as social deprivation, psychiatric diagnosis, prescriptions for antidepressants/antipsychotics, and alcohol use yielded better predictive risk models than the Framingham Risk Score** for adults with CMI

BOTTOM LINE: "Standard" diabetes interventions and/or a focus entirely on biological variables may fail to account for key factors that contribute to DMI complications among those with CMI. This has important clinical care implications

AHRQ: effectivehealthcare.ahrq.gov/ehc/products/377/1464/mental-illness-cardio-risk-executive-130422.pdf.

D'Agostino RB, et al, Circulation. 2008

Osborn DP, et al, JAMA Psychiatry. 72(2):143-51.2015

Antipsychotic drugs and DM risk in CMI

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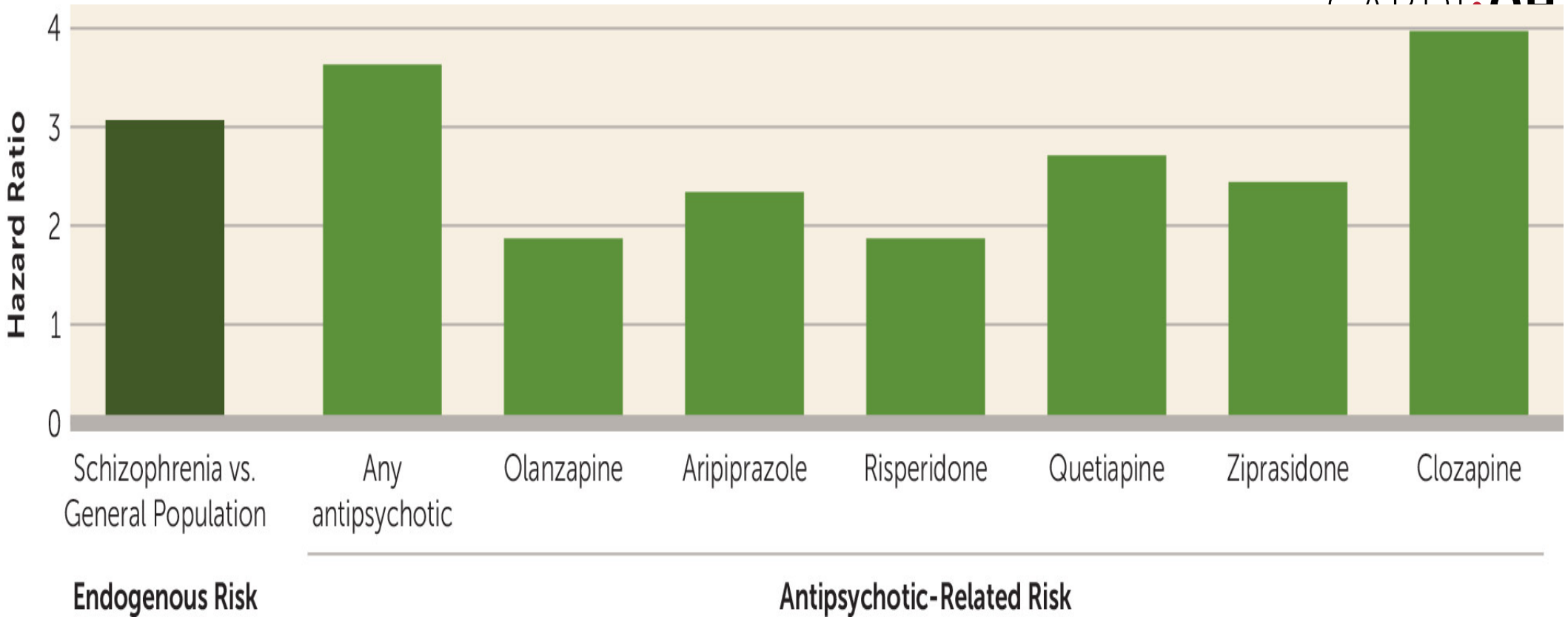
- About 12% of people receiving antipsychotic drugs have DM
- Meta-analyses suggest the prevalence of DM is not appreciably increased in drug-naïve, first-episode CMI patients.
- Many (but not all) studies suggest second-generation antipsychotics (SGAs) have greater diabetogenic potential vs. first-generation antipsychotics (FGAs)
- Metabolic abnormalities accumulate rapidly after the initiation of treatment
- Antipsychotics may induce DM independent of adiposity, contributing to DM both indirectly, by inducing weight gain, and directly, by promoting insulin resistance
- Children and youth exposed to antipsychotics have a 3-fold increased risk of DM

Chesney E, et al World Psychiatry. 2014 Jun; 13(2):153-60.

Mitchell AJ, et al,Schizophr Bull. 2013 Mar; 39(2):295-305

Correll, C. U., et al, *World Psychiatry*, *14*(2), 119–136. 2015



Andreasson, Am J Psychiatry2017https://doi.org/10.1176/appi.ajp.2017.17040409

Antidepressant drugs and DM risk in CMI

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- A meta-analysis found that antidepressants increased the likelihood of new-onset DM (OR = 1.50, 95% CI: 1.08-2.10; HR = 1.19, 95% CI: 1.08-1.32). However, because only observational studies were included in this analysis, a causal relationship could not be established
- Increased DM risk may be associated with use of tricyclic antidepressants (TCAs) and selective serotonin reuptake inhibitors (SSRIs) (OR = 1.89)
- *DM risk with antidepressants might be elevated with long-term use of TCA or SSRIs and/or in high-risk patients*

Bhattacharjee S, et al, Diabetes Metab Res Rev. 29(4):273-84.2013

Brown LC, et al, Diabetes Res Clin Pract. 79(1):61-7.2008

Rubin RR, et al Diabetes Care. 2008 Mar; 31(3):420-6.

Supporting adults with comorbid DM & CMI

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- Standard diabetes education needs to accommodate possible cognitive deficits and/or significant mood states that may make knowledge accumulation and retention challenging
- Prescribe psychotropic drugs that minimize metabolic/weight gain propensity
- Optimize outcomes of psychiatric comorbidity
- Self-management support that addresses bio-behavioral factors
- Collaborative or integrated care models that include behavioral medicine

Anamalie. Int J Endocrinology. Volume 2015 |Article ID 969182 |

https://doi.org/10.1155/2015/969182

https://store.samhsa.gov/sites/default/files/d7/priv/sma13-4780.pdf

Adverse effects of psychotropic drugs on physical health outcomes

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Physical condition	Antipsychotics	Antidepressants	Mood stabilizers
Obesity	**0/+ (haloperidol, lurasidone, ziprasidone, aripiprazole) to +++ (clozapine, olanzapine, low potency FGAs)**	**– (bupropion) to + (mirtazapine, paroxetine, TCAs)**	**0 (lamotrigine) to ++ (valproate, lithium)**
Dyslipidemia	+ to ++	0 to + (if weight gain)	– (valproate: cholesterol) to +
Diabetes	**0/+ (haloperidol, lurasidone, ziprasidone, aripiprazole) to +++ (clozapine and olanzapine > low and mid potency FGAs)**	**0 to +**	**0 to ++ (valproate)**
Hypertension	0 to ++	0 to + (venlafaxine)	0

– = reduction; 0 = likely/generally no effect; + = some effect; ++ = moderate effect; +++ = marked effect, ? = questionable
FGAs – first-generation antipsychotics, SSRIs – selective serotonin reuptake inhibitors, TCAs – tricyclic antidepressants

Adapted from Correll, C. U., et al, *World Psychiatry*, *14*(2), 119–136. 2015

Minimizing metabolic liability with antipsychotic & antidepressant drugs

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- Young, drug–naïve patients are particularly vulnerable to weight gain
- Use SGAs with high metabolic liability (quetiapine,etc.) conservatively and limit off–label use
- Patients should be screened before drug initiation and monitored subsequently following standard guidelines, such as those provided by the ADA
- Patients with significant weight gain should be switched to a lower metabolic liability SGA.
- Metformin may help young patients with limited exposure to antipsychotic drugs if lifestyle interventions fail and switching the SGA is not an option. However, benefits may be modest
- For second-generation antidepressants (SGADs), paroxetine and mirtazapine are associated with weight gain
- Bupropion may cause modest weight loss.
- Other SGADs are mostly weight neutral, but individual variations may occur.

Hasnain M, Vieweg WV, Hollett B. Postgrad Med. 2012 Jul; 124(4):154-67

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Table 1 – Metabolic monitoring parameters based on American Diabetes Association/ American Psychiatric Association consensus guidelines[4]						
	Baseline	**Week 4**	**Week 8**	**Week 12**	**Every 3 months thereafter**	**Annually**
Medical history[a]	X			X		X
Weight (BMI)	X	X	X	X	X	X
Waist circumference	X			X		X
Blood pressure	X			X		X
Fasting glucose/hemoglobin A_{1c}	X			X		X
Fasting lipids	X			X		X

[a] Personal and family history of obesity, diabetes, hypertension, and cardiovascular disease.

Diabetes Care. 2004;27:596-601.

Piette and Kerr DM/Chronic disease comorbidity model

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- Concordant comorbidity has similar pathogenesis/management (ex. Mental illness & DM)
- Discordant comorbidty has different pathogenesis/management (ex. Mental illness & musculoskeletal disorder)
- Dominant comorbidity (ex. Metastatic cancer) overshadows all other illnesses
- Concordant conditions may share underlying pathology such as inflammation. This may help in identifying "2-fer" treatments such as stress-management
- Identifying discordant comorbidity is essential and targeted approaches to comorbidity can improve both CMI and DM

Piette JD, Kerr EA. Diabetes Care 2006;29:725–731

*Pentakota Diabetes Care **June 2012** vol. 35 no. 6*

Managing psychiatric comorbidity to optimize DM control

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- 2006-2010 National Surveys on Drug Use and Health, found > 1/3 of people with DM age 18 -25 had past-month binge drinking, putting them at risk for DM complications.
- DM self-care behaviors are inversely correlated with alcohol consumption; rates of non-adherence among those who drink are ↑ starting at just 1 drink/day vs. abstinent patients. Treatment of substance use disorders may improve DM control.
- Depression predisposes to weight gain and is a risk factor for glucose dysregulation. Optimal treatment of depression improves glucose metabolism and makes it easier for patients to manage complex self-management plans.

Center for Behavioral Health Statistics and Quality. (2012, March 27). Substance Abuse and Mental Health Services Administration.

Ahmed, A. T., et al Diabetic Medicine, 23(7), 795–802.2006

Hasnain M, Vieweg WV, Hollett B. Postgrad Med. 2012 Jul; 124(4):154-67

Enhancing medical care with self-management/multi-component support

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- Chronic disease self-management (CDSM) is an evidence-based set of practices that are effective in improving the health of people with multiple morbidities
- CDSM approaches are acceptable to those with CMI
- Incorporating CDSM practices into standard clinical care may be a way to enhance uptake and impact
- nutrition and exercise counselling, behavioral modelling and increased disease awareness aiming to reduce HbA1c, fasting plasma glucose, BMI and weight can collectively show modest positive impact Lorig , Ann Behav Med 2003;26(1):1–7. , Chodosh, Ann Intern Med 2005;143:427–38, Gron et al. Primary Care Diabetes 2018DOI:https://doi.org/10.1016/j.pcd.2018.03.008

The Effectiveness of Pharmacological and Non-Pharmacological Interventions for Improving Glycaemic Control in Adults with Severe Mental Illness: A Systematic Review and Meta-Analysis

Taylor et al. *PloS one* vol. 12,1 e0168549. 5. 2017

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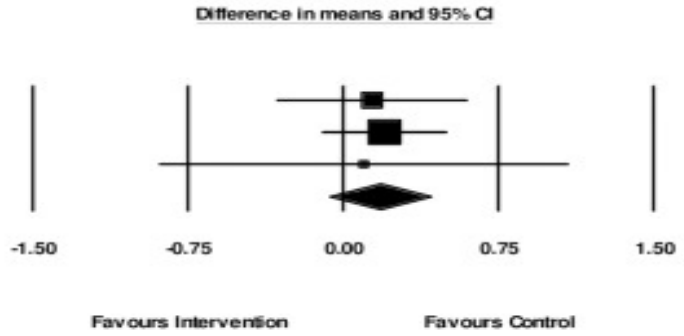
HbA_{1c}

Study name	Subgroup within study	Outcome	Statistics for each study: Difference in means	Standard error	Lower limit	Upper limit	Sample size: Intervention	Control	Relative weight
Forsberg 2008	Behavioural	HbA1c	0.140	0.236	-0.322	0.602	21	12	28.14
Gillhoff 2010	Behavioural	HbA1c	0.200	0.154	-0.102	0.502	26	24	65.73
McKibbin 2006	Behavioural	HbA1c	0.100	0.505	-0.890	1.090	28	29	6.13
			0.177	0.125	-0.068	0.422			

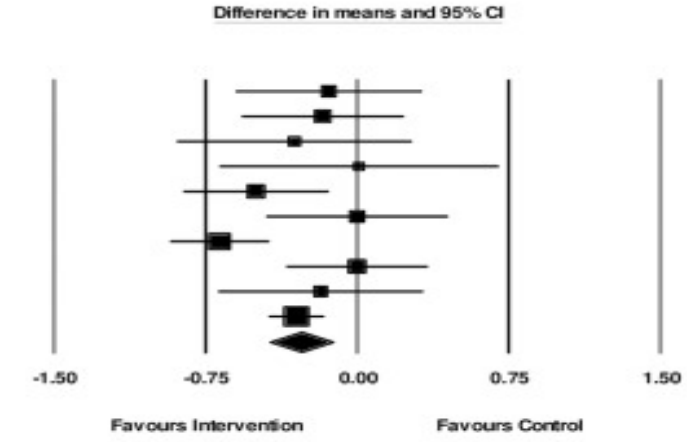
Difference in means = 0.177% (95% confidence interval = -0.068 to 0.422)
Test of significance: Z = 1.415, p = 0.16; Test of heterogeneity: I^2 = 0.00, p = 0.97



Fasting blood glucose

Study name	Subgroup within study	Outcome	Statistics for each study: Difference in means	Standard error	Lower limit	Upper limit	Sample size: Intervention	Control	Relative weight
Attux 2013	Behavioural	Fasting glucose	-0.140	0.235	-0.601	0.321	60	66	7.82
Cordes 2014	Behavioural	Fasting glucose	-0.170	0.205	-0.572	0.232	13	18	9.37
Daumit 2013	Behavioural	Fasting glucose	-0.310	0.297	-0.891	0.271	96	95	5.56
Forsberg 2008	Behavioural	Fasting glucose	0.010	0.352	-0.681	0.701	23	14	4.21
Green 2015	Behavioural	Fasting glucose	-0.500	0.183	-0.859	-0.141	104	96	10.71
Mauri 2008	Behavioural	Fasting glucose	0.000	0.230	-0.450	0.450	15	18	8.08
Poulin 2007	Behavioural	Fasting glucose	-0.680	0.125	-0.925	-0.435	59	51	15.42
Scheewe 2013	Behavioural	Fasting glucose	0.000	0.179	-0.351	0.351	29	25	11.01
Wu 2007	Behavioural	Fasting glucose	-0.180	0.259	-0.687	0.327	28	25	6.83
Wu 2008A	Behavioural	Fasting glucose	-0.300	0.070	-0.437	-0.163	32	32	20.98
			-0.277	0.079	-0.433	-0.122			

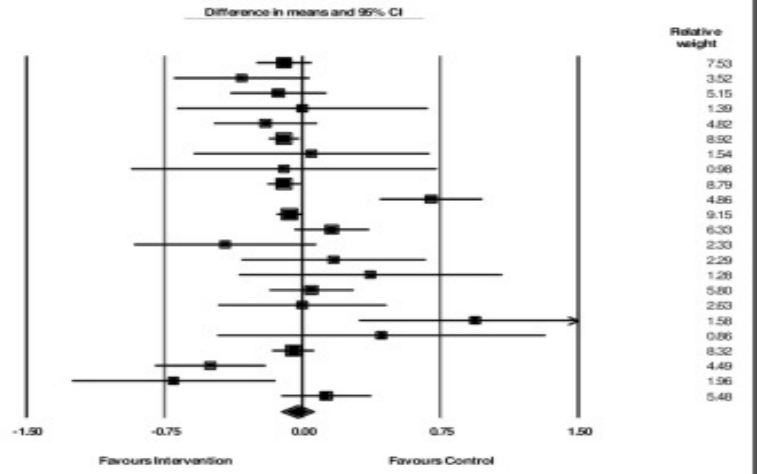
Difference in means = -0.277mmol/L (95% confidence interval = -0.433 to -0.122)
Test of significance: Z = -3.500, p < 0.001*; Test of heterogeneity: I^2 = 46.147, p = 0.05



HbA_{1c}

Study name	Subgroup within study	Outcome	Statistics for each study: Difference in means	Standard error	Lower limit	Upper limit	Sample size: Intervention	Control	Relative weight
Deberdt 2008	Anti-psychotic switching	HbA1c	-0.100	0.077	-0.251	0.051	65	68	7.53
Fan 2013	Anti-psychotic switching	HbA1c	-0.300	0.189	-0.700	0.040	16	14	3.52
Henderson 2009 A	Anti-psychotic switching	HbA1c	-0.130	0.134	-0.392	0.132	8	7	5.15
Kanagianis 2009	Anti-psychotic switching	HbA1c	0.000	0.348	-0.682	0.682	84	65	1.39
Kusumi 2012	Anti-psychotic switching	HbA1c	-0.200	0.143	-0.481	0.081	61	57	4.82
Stroup 2011	Anti-psychotic switching	HbA1c	-0.100	0.043	-0.183	-0.017	89	98	8.92
Baptista 2007	Diabetes medication	HbA1c	0.050	0.327	-0.592	0.692	33	31	1.54
Baptista 2009	Diabetes medication	HbA1c	-0.100	0.424	-0.931	0.731	13	14	0.98
Carrizo 2009	Diabetes medication	HbA1c	-0.100	0.046	-0.191	-0.009	24	30	8.79
Henderson 2009 B	Diabetes medication	HbA1c	0.700	0.142	0.421	0.979	8	10	4.86
Jarskog 2013	Diabetes medication	HbA1c	-0.070	0.036	-0.140	-0.000	75	71	9.15
Li 2013	Diabetes medication	HbA1c	0.160	0.104	-0.044	0.364	18	21	6.33
Smith 2013 China	Diabetes medication	HbA1c	-0.420	0.253	-0.916	0.076	5	5	2.33
Smith 2013 US	Diabetes medication	HbA1c	0.170	0.256	-0.332	0.672	25	19	2.29
Borba 2011	Other	HbA1c	0.370	0.365	-0.346	1.086	14	6	1.28
Fadai 2014	Other	HbA1c	0.050	0.117	-0.179	0.279	40	21	5.80
Lee 2013	Other	HbA1c	-0.000	0.234	-0.458	0.458	48	35	2.63
Tek 2014	Other	HbA1c	0.940	0.323	0.308	1.572	11	12	1.58
Baptista 2008	Weight loss and diabetes combination	HbA1c	0.430	0.456	-0.463	1.323	13	15	0.86
Hoffmann 2012	Weight loss and diabetes combination	HbA1c	-0.050	0.058	-0.164	0.064	149	50	8.32
Biedermann 2014	Weight loss medication	HbA1c	-0.500	0.154	-0.801	-0.199	5	6	4.49
Henderson 2005	Weight loss medication	HbA1c	-0.700	0.283	-1.254	-0.146	19	18	1.96
Henderson 2007	Weight loss medication	HbA1c	0.130	0.125	-0.115	0.375	10	8	5.48
			-0.029	0.044	-0.115	0.058			

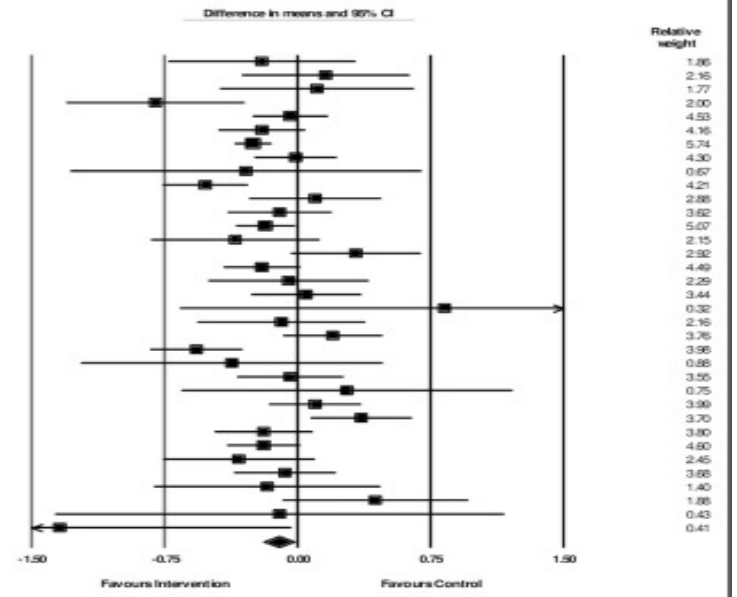
Difference in means = -0.029% (95% confidence interval = -0.115 to 0.058)
Test of significance: Z = -0.645, p = 0.52; Test of heterogeneity: I^2 = 68.577, p < 0.001*



Fasting blood glucose

Study name	Subgroup within study	Outcome	Statistics for each study: Difference in means	Standard error	Lower limit	Upper limit	Sample size: Intervention	Control	Relative weight
Baptista 2006	Metformin	Fasting glucose	-0.200	0.269	-0.728	0.328	19	18	1.86
Baptista 2007	Metformin	Fasting glucose	0.160	0.241	-0.313	0.633	36	36	2.16
Carrizo 2009	Metformin	Fasting glucose	0.110	0.279	-0.437	0.657	24	30	1.77
Chen 2013	Metformin	Fasting glucose	-0.800	0.256	-1.302	-0.298	28	27	2.00
Jarskog 2013	Metformin	Fasting glucose	-0.040	0.108	-0.253	0.173	75	71	4.53
Wang 2012	Metformin	Fasting glucose	-0.200	0.125	-0.445	0.045	32	34	4.16
Wu 2008 A	Metformin	Fasting glucose	-0.250	0.053	-0.354	-0.146	64	64	5.74
Wu 2008 B	Metformin	Fasting glucose	-0.010	0.119	-0.242	0.222	18	19	4.30
Borba 2011	Other	Fasting glucose	-0.290	0.505	-1.280	0.700	14	6	0.67
Fadai 2014	Other	Fasting glucose	-0.520	0.123	-0.760	-0.280	40	21	4.21
Hollie-Pickersle 2015	Other	Fasting glucose	0.100	0.190	-0.272	0.472	23	22	2.88
Lee 2013	Other	Fasting glucose	-0.100	0.150	-0.394	0.194	48	35	3.62
Lu 2004	Other	Fasting glucose	-0.180	0.085	-0.347	-0.013	34	34	5.07
Modabbernia 2014	Other	Fasting glucose	-0.350	0.243	-0.826	0.126	18	18	2.15
Baptista 2008	Weight loss and diabetes combination	Fasting glucose	0.330	0.188	-0.038	0.698	13	15	2.92
Hoffmann 2012	Weight loss and diabetes combination	Fasting glucose	-0.200	0.110	-0.416	0.016	149	50	4.49
Deberdt 2008	Anti-psychotic switching	Fasting glucose	-0.050	0.231	-0.502	0.402	65	68	2.29
Amami-Wauzman 2013	Weight loss medication	Fasting glucose	0.050	0.159	-0.261	0.361	25	29	3.44
Henderson 2007	Weight loss medication	Fasting glucose	0.830	0.781	-0.692	2.352	10	8	0.32
Joffe 2008	Weight loss medication	Fasting glucose	-0.090	0.242	-0.564	0.384	31	32	2.16
McElroy 2012	Weight loss medication	Fasting glucose	0.200	0.143	-0.081	0.481	20	22	3.76
Narula 2010	Weight loss medication	Fasting glucose	-0.570	0.133	-0.830	-0.310	33	34	3.98
Fan 2013	Anti-psychotic switching	Fasting glucose	-0.370	0.434	-1.221	0.481	16	14	0.88
Fleischhacker 2010	Anti-psychotic switching	Fasting glucose	-0.040	0.153	-0.340	0.260	96	87	3.55
Henderson 2009 A	Anti-psychotic switching	Fasting glucose	0.280	0.477	-0.654	1.214	8	7	0.75
Kanagianis 2009	Anti-psychotic switching	Fasting glucose	0.100	0.132	-0.160	0.360	84	65	3.99
Kusumi 2012	Anti-psychotic switching	Fasting glucose	0.360	0.146	0.074	0.646	61	57	3.70
Newcomer 2008	Anti-psychotic switching	Fasting glucose	-0.190	0.141	-0.466	0.086	80	76	3.80
Stroup 2011	Anti-psychotic switching	Fasting glucose	-0.190	0.105	-0.396	0.016	89	98	4.60
Wani 2015	Anti-psychotic switching	Fasting glucose	-0.330	0.219	-0.759	0.099	21	26	2.45
Baptista 2009	Diabetes medication	Fasting glucose	-0.070	0.147	-0.358	0.218	13	14	3.68
Henderson 2009 B	Diabetes medication	Fasting glucose	-0.170	0.326	-0.809	0.469	8	10	1.40
Li 2013	Diabetes medication	Fasting glucose	0.440	0.257	-0.064	0.964	18	21	1.88
Smith 2013 China	Diabetes medication	Fasting glucose	-0.100	0.646	-1.367	1.167	5	5	0.43
Smith 2013 US	Diabetes medication	Fasting glucose	-1.340	0.668	-2.646	-0.034	25	19	0.41
			-0.106	0.044	-0.191	-0.020	1343	1193	

Difference in means = -0.106mmol/L (95% confidence interval = -0.191 to -0.020)
Test of significance: Z = -2.407, p = 0.02*; Test of heterogeneity: I^2 = 57.352, p < 0.001*



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Conclusions:

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- People with CMI are a special population with more difficult DM management challenges
- Effective management includes:

- Minimizing metabolic liability of psychotropic drugs
- Prevention/self-management support for risk factor management (especially bio-behavioral factors!)
- Coordination between primary care and behavioral care providers
- Engaging patients in care that is long-term & sustaintable

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Thank you!

Questions/Discussion