



Morita therapy

Introduction

Morita therapy is a treatment approach developed by Shoma Morita and is most commonly used in some Asian countries, including Japan and China. Morita therapy focuses on mental health from a collective perspective, rather than the perspective of the individual¹, removing the preoccupation with symptoms and neuroticism and instead focusing on constructive behaviours. While some Morita therapy programs have been updated and shortened (~4 weeks), the original Morita therapy guideline is aimed at inpatients and is divided into four phases:

1. Seven days of isolated bed rest, with no access to any form of entertainment.
2. Four to seven days of light work (graded activity) within the treatment facility, in addition to monitored diary writing and therapist appointments, where the therapist pays strategic inattention to symptoms, and uses contingency management to focus on daily activities.
3. A longer period of work (1-2 months) with increasing engagement in more demanding tasks within the treatment facility, and gradual collaboration with other patients.
4. Preparation for daily living outside the treatment facility (1-4 weeks), which may include commuting to school or work from the facility.

Method

We have included only systematic reviews (systematic literature search, detailed methodology with inclusion/exclusion criteria) published in full text, in English, from the year 2000 that report results separately for people with a diagnosis of schizophrenia, schizoaffective disorder, schizophreniform disorder or first episode schizophrenia.

Reviews were identified by searching MEDLINE, EMBASE, CINAHL, Current Contents, PsycINFO and the Cochrane Library databases. Hand searching reference lists of identified reviews was also conducted. When multiple copies of reviews were found, only the most recent version was included. Reviews with pooled data are prioritised for inclusion.

Review reporting assessment was guided by the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) checklist that describes a preferred way to present a meta-analysis². Reviews rated as having less than 50% of items checked have been excluded from the library. The PRISMA flow diagram is a suggested way of providing information about studies included and excluded with reasons for exclusion. Where no flow diagram has been presented by individual reviews, but identified studies have been described in the text, reviews have been checked for this item. Note that early reviews may have been guided by less stringent reporting checklists than the PRISMA, and that some reviews may have been limited by journal guidelines.

Evidence was graded using the Grading of Recommendations Assessment, Development and Evaluation (GRADE) Working Group approach where high quality evidence such as that gained from randomised controlled trials (RCTs) may be downgraded to moderate or low if review and study quality is limited, if there is inconsistency in results, indirect comparisons, imprecise or sparse data and high probability of reporting bias. It may also be downgraded if risks associated with the intervention or other matter under review are high. Conversely, low quality evidence such as that gained from observational studies may be upgraded if effect sizes are large or if there is a dose dependent response. We have also taken into account sample size and whether results are consistent, precise and direct with low associated risks



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(see end of table for an explanation of these terms)³. The resulting table represents an objective summary of the available evidence, although the conclusions are solely the opinion of staff of NeuRA (Neuroscience Research Australia).

Results

We found one systematic review that met our inclusion criteria¹.

- When compared to standard care, moderate to low quality evidence suggests improved negative symptoms (measured by SANS endpoint scores) with Morita therapy in the short term. Low quality evidence is unable to determine the benefits of Morita therapy over standard care for other symptoms or for functioning.
- When compared to rehabilitation, moderate quality evidence suggests Morita therapy may be beneficial for symptoms (measured by BPRS endpoint scores), insight and general functioning.



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Morita therapy for schizophrenia

Cochrane Database of Systematic Reviews 2007; (1): CD006346

[View review abstract online](#)

Comparison 1	Morita therapy plus standard care vs. standard care. Treatment duration ranged from 6 weeks to 6 months.
Summary of evidence	Moderate quality evidence (medium to large samples, consistent, direct, imprecise) suggests no benefit of Morita therapy over standard care for study retention. Moderate to low quality evidence (medium-sized samples, inconsistent, unable to assess precision, direct) suggests improved negative symptoms (measured by SANS endpoint scores) with Morita therapy in the short term. Low quality evidence (small samples) is unable to determine the benefits of Morita therapy for other symptoms or for functioning.
Study retention	
<p style="text-align: center;"><i>No significant differences between groups in study attrition for any reason;</i></p> <p>Short term (< 12 weeks): 6 RCTs, N = 475, RR = 0.67, 95%CI 0.11 to 3.96, $p = 0.66$, $Q = 0.14$, $p = 0.71$, $I^2 = 0\%$</p> <p>Medium term (13-52 weeks): 4 RCTs, N = 286, RR = 1.25, 95%CI 0.36 to 4.43, $p = 0.73$, $Q = 0.0$, $p = 1.00$, $I^2 = 0\%$</p> <p style="text-align: center;"><i>No significant differences between groups in study attrition for specific reasons;</i></p> <p>Discharge, short term: 2 RCTs, N = 132, RR = 0.43, 95%CI 0.07 to 2.84, $p = 0.38$, $Q = 0.04$, $p = 0.84$, $I^2 = 0\%$</p> <p>Discharge, medium term: 1 RCT, N = 120, RR = 0.33, 95%CI 0.01 to 8.02, $p = 0.50$</p> <p>Relapse, short term: 1 RCT, N = 82, RR = 3.00, 95%CI 0.13 to 71.56, $p = 0.50$</p> <p>Uncooperative, short term: 1 RCT, N = 120, RR = 3.00, 95%CI 0.32 to 28.03, $p = 0.34$</p> <p>Lost to follow up: 1 RCT, N = 120, RR = 1.00, 95%CI 0.15 to 6.87, $p = 1.00$</p>	
Mental state	
<p>Overall symptoms</p> <p><i>BPRS clinically important improvement ratings (total score <25-30% = no improvement) were</i></p>	



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significantly better with Morita therapy in the short and the medium term;

Short term: 1 RCT, N = 66, RR = 0.36, 95%CI 0.13 to 1.03, $p = 0.056$

Medium term: 1 RCT, N = 76, RR = 0.36, 95%CI 0.14 to 0.89, $p = 0.028$

BPRS endpoint scores were significantly better with Morita therapy in the medium term, but not in the short term;

Short term: 3 RCTs, N = 189, WMD = -4.33, 95%CI -10.28 to 1.62, $p = 0.15$, $Q = 32.94$, $p < 0.0001$, $I^2 = 94\%$

Medium term: 2 RCTs, N = 124, WMD = -5.19, 95%CI -9.64 to -0.74, $p = 0.022$, $Q = 5.77$, $p = 0.02$, $I^2 = 83\%$

BPRS % change scores were significantly better with Morita therapy in the short term, but not in the medium term;

Short term: 1 RCT, N = 100, WMD = 3.90, 95%CI 0.55 to 7.25, $p = 0.022$

Medium term: 1 RCT, N = 111, WMD = 4.46, 95%CI -2.26 to 11.18, $p = 0.19$

Negative symptoms

SANS clinically important improvement ratings were significantly better with Morita therapy in the medium term, but not in the short term;

Short term: 1 RCT, N = 50, RR = 0.89, 95%CI 0.41 to 1.93, $p = 0.77$

Medium term: 1 RCT, N = 42, RR = 0.25, 95%CI 0.07 to 0.76, $p = 0.014$

SANS endpoint scores were significantly better with Morita therapy in the short and the medium term;

Short term: 4 RCTs, N = 323, WMD = -12.94, 95%CI -21.57 to -4.32, $p = 0.0033$, $Q = 98.54$, $p < 0.0001$, $I^2 = 97\%$

Medium term: 1 RCT, N = 76, WMD = -4.57, 95%CI -8.56 to -0.58, $p = 0.025$

Depression symptoms

HAM-D clinically important improvement ratings showed a trend effect of greater improvement with Morita therapy in the short term;

1 RCT, N = 104, RR = 0.36, 95%CI 0.12 to 1.07, $p = 0.066$

HAM-D endpoint scores were significantly better with Morita therapy in the short term;

1 RCT, N = 104, WMD = -3.59, 95%CI -5.64 to -1.54, $p = 0.0061$

Functioning



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<p><i>General functioning was significantly better with Morita therapy in the short and medium term;</i> Activities of daily living scale, short term: 1 RCT, N = 104, WMD = -4.14, 95%CI -7.67 to -0.61, $p = 0.022$ Activities of daily living scale, medium term: 1 RCT, N = 48, WMD = -10.50, 95%CI -12.50 to -8.50, $p < 0.0001$ Inpatient psychiatric rehabilitation outcome scale, short term: 1 RCT, N = 80, WMD = -45.87, 95%CI -50.68 to -41.06, $p < 0.0001$ Nurses observation scale for inpatient evaluation, short term: 1 RCT, N = 66, WMD = 15.20, 95%CI 9.00 to 21.40, $p < 0.0001$</p> <p><i>Social functioning was significantly better with Morita therapy in the medium term;</i> Social disability screening schedule: 1 RCT, N = 111, WMD = 16.40, 95%CI 9.71 to 23.09, $p < 0.0001$</p>	
Risks	No significant differences were found between Morita therapy and standard care for risk of adverse events, including somnolence (N = 104, RR = 0.86, 95%CI 0.31, 2.38, $p = 0.77$), postural hypertension (N = 104, RR = 1.33, 95%CI 0.31, 5.67, $p = 0.70$), and treatment emergent symptoms (TESS) endpoint scores (N = 104, WMD = -0.18, 95%CI -0.90, 0.54, $p = 0.63$).
Consistency in results	Consistent for study retention, otherwise results are inconsistent or not applicable (1 RCT).
Precision in results	Imprecise for study retention, unable to assess mental state/social function (WMDs are not standardised).
Directness of results	Direct
Comparison 2	Morita therapy plus standard care vs. rehabilitation plus standard care. Treatment duration = 6 weeks.
Summary of evidence	Moderate quality evidence (medium-sized sample, consistent, direct, imprecise) suggests Morita therapy may be beneficial for symptoms (measured by BPRS endpoint scores), insight and general functioning, with no benefit over rehabilitation for study retention.
Study attrition	



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<p><i>No significant differences between groups in study attrition for any reason;</i> Short term: 2 RCTs, N = 302, RR = 1.00, 95%CI 0.47 to 2.11, $p = 1.0$, $Q = 0.00$, $p = 1.0$, $I^2 = 0\%$</p>	
<p>Mental state</p>	
<p><i>No significant differences between groups in BPRS clinically important improvement ratings in the short term;</i> Short term: 1 RCT, N = 120, RR = 0.89, 95%CI 0.52 to 1.55, $p = 0.69$ <i>BPRS endpoint scores were significantly better with Morita therapy in the short term;</i> Short term: 2 RCTs, N = 278, WMD = -6.95, 95%CI -9.26 to -4.64, $p < 0.0001$, $Q = 0.0$, $p = 0.99$, $I^2 = 0\%$</p>	
<p>Insight and functioning</p>	
<p><i>Insight endpoint scores (clinician judgement) were significantly better with Morita therapy in the short term;</i> Short term: 2 RCTs, N = 278, WMD = -1.11, 95%CI -1.32 to -0.91, $p < 0.0001$, $Q = 0.0$, $p = 0.96$, $I^2 = 0\%$ <i>General functioning endpoint scores were significantly better with Morita therapy in the short term;</i> Inpatient psychiatric rehabilitation outcome scale, short term: 2 RCTs, N = 278, WMD = -18;14, 95%CI -21.33 to -14.95, $p < 0.0001$, $Q = 0.0$, $p = 0.96$, $I^2 = 0\%$</p>	
Risks	No adverse effects are reported.
Consistency in results	Consistent where applicable (>1 RCT).
Precision in results	Imprecise for RRs, unable to assess WMDs.
Directness of results	Direct

Explanation of acronyms

BPRS = Brief Psychiatric Rating Scale, CI = Confidence Interval, HAM-D = Hamilton Rating Scale for Depression, I^2 = the percentage of the variability in effect estimates that is due to heterogeneity rather than sampling error (chance), N = number of participants, p = statistical probability of obtaining that result ($p < 0.05$ generally regarded as significant), Q = Q statistic for the test of heterogeneity, RCT = randomised controlled trial, RR = relative risk, SANS = Scale for the Assessment of Negative Symptoms, vs = versus, WMD = weighted mean difference



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Explanation of technical terms

* Bias has the potential to affect reviews of both RCT and observational studies. Forms of bias include; reporting bias – selective reporting of results; publication bias - trials that are not formally published tend to show less effect than published trials, further if there are statistically significant differences between groups in a trial, these trial results tend to get published before those of trials without significant differences; language bias – only including English language reports; funding bias - source of funding for the primary research with selective reporting of results within primary studies; outcome variable selection bias; database bias - including reports from some databases and not others; citation bias - preferential citation of authors. Trials can also be subject to bias when evaluators are not blind to treatment condition and selection bias of participants if trial samples are small⁴.

† Different effect measures are reported by different reviews.

Prevalence refers to how many existing cases there are at a particular point in time. Incidence refers to how many new cases there are per population in a specified time period. Incidence is usually reported as the number of new cases per 100,000 people per year. Alternatively some studies present the number of new cases that have accumulated over several years against a person-years denominator. This denominator is the sum of individual units of time that the persons in the population are at risk of becoming a case. It takes into account the size of the underlying population sample and its age structure over the duration of observation.

Reliability and validity refers to how accurate the instrument is. Sensitivity is the proportion of actual positives that are correctly identified (100% sensitivity = correct identification of all actual positives) and specificity is the proportion of negatives that are correctly identified (100% specificity = not identifying anyone as positive if they are truly not).

Weighted mean difference scores refer to mean differences between treatment and comparison groups after treatment (or occasionally pre to post treatment) and in a randomised trial there is an assumption that both groups are comparable on this measure prior to treatment. Standardised mean differences are divided by the pooled standard deviation (or the standard deviation of one group when groups are homogenous), which allows results from different scales to be combined and compared. Each study's mean difference is then given a weighting depending on the size of the sample and the variability in the data. Less than 0.4 represents a small effect, around 0.5 a medium effect, and over 0.8 represents a large effect⁴.

Odds ratio (OR) or relative risk (RR) refers to the probability of a reduction (< 1) or an increase (> 1) in a particular outcome in a treatment group, or a group exposed to a risk factor, relative to the comparison group. For example, a RR of 0.75 translates to a reduction in risk of an outcome of 25% relative to those not receiving the treatment or not exposed to the risk factor. Conversely, a RR of 1.25 translates to an increased risk of 25% relative to those not receiving treatment or not having been exposed to a risk factor. A RR or OR of 1.00 means there is no difference between groups. A medium effect is considered if $RR > 2$ or < 0.5 and a large effect if $RR > 5$ or < 0.2 ⁵. lnOR stands for logarithmic OR where a lnOR of 0 shows no difference between groups. Hazard ratios



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measure the effect of an explanatory variable on the hazard or risk of an event.

Correlation coefficients (eg, r) indicate the strength of association or relationship between variables. They can provide an indirect indication of prediction, but do not confirm causality due to possible and often unforeseen confounding variables. An r of 0.10 represents a weak association, 0.25 a medium association and 0.40 and over represents a strong association. Unstandardised (b) regression coefficients indicate the average change in the dependent variable associated with a 1 unit change in the independent variable, statistically controlling for the other independent variables. Standardised regression coefficients represent the change being in units of standard deviations to allow comparison across different scales.

‡ Inconsistency refers to differing estimates of effect across studies (i.e. heterogeneity or variability in results) that is not explained by subgroup analyses and therefore reduces confidence in the effect estimate. I^2 is the percentage of the variability in effect estimates that is due to heterogeneity rather than sampling error (chance) - 0% to 40%: heterogeneity might not be important, 30% to 60%: may represent moderate heterogeneity, 50% to 90%: may represent considerable heterogeneity and over this is considerable heterogeneity. I^2 can be calculated from Q (chi-square) for the test of heterogeneity with the following formula⁴;

$$I^2 = \left(\frac{Q - df}{Q} \right) \times 100\%$$

§ Imprecision refers to wide confidence intervals indicating a lack of confidence in the effect estimate. Based on GRADE recommendations, a result for continuous data (standardised mean differences, not weighted mean differences) is considered imprecise if the upper or lower confidence limit crosses an effect size of 0.5 in either direction, and for binary and correlation data, an effect size of 0.25. GRADE also recommends downgrading the evidence when sample size is smaller than 300 (for binary data) and 400 (for continuous data), although for some topics, these criteria should be relaxed⁶.

|| Indirectness of comparison occurs when a comparison of intervention A versus B is not available but A was compared with C and B was compared with C that allows indirect comparisons of the magnitude of effect of A versus B. Indirectness of population, comparator and/or outcome can also occur when the available evidence regarding a particular population, intervention, comparator, or outcome is not available and is therefore inferred from available evidence. These inferred treatment effect sizes are of lower quality than those gained from head-to-head comparisons of A and B.



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References

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