

Using Fixed and Relative Optimal Discriminant Thresholds in Randomized Blocks (Matched-Pairs) Designs

Paul R. Yarnold, Ph.D. and Ariel Linden, Dr.P.H.

Optimal Data Analysis, LLC

Linden Consulting Group, LLC

Optimal discriminant analysis (ODA) is often used to compare values of one (or more) attributes between two (or more) groups of observations with respect to a *fixed* discriminant threshold that maximizes accuracy normed against chance for the sample.¹⁻²⁹ However, a recent study using a matched-pairs design found that using a *relative* discriminant threshold to assess an (exploratory or confirmatory) *a priori* hypothesis *separately for each pair* of observations can identify inter-group differences which otherwise are too subtle to be identified by using fixed thresholds.³⁰ The present investigation replicates the finding regarding efficacy of relative thresholds for matched-pairs designs, this time for a randomized blocks design consisting of two patient groups (one group assigned to take an antidepressant drug, the other group assigned to take a placebo) between which a numerical measure of depression was compared.³¹ Several recommendations are made concerning use of improved modern optimal statistical alternatives for this class of experimental design.

Fleiss presents an example of a randomized blocks (matched-pairs) experiment comparing scores on a depression measure for 60 patients formed into 30 pairs matched on gender, age (within one decade for females, or two decades for males), and time of study entry (within one month).³¹ Data are given by pair in Table 1: the first value in each row is score on the Hamilton depression scale (higher scores indicate worse depression) for the patient in the *Imipramine* condition; the second value is the depression score for the paired patient in the *Placebo* con-

dition; and the third value is the difference between these scores—a negative difference indicates the patient in the Placebo condition has a higher depression score (is more depressed) than the patient in the Imipramine condition). In the Table, the pairs were first sorted in order of increasing depression score for the patient in the Imipramine condition; for tied scores, data were sorted a second time by increasing depression score for the patient in the Placebo condition.

Table 1: Hamilton Depression Scale Scores of 60 Patients in 30 Matched Pairs³¹

Group		I-P	Relative Threshold	
Imipramine	Placebo		I≤P	I<P
3	3	0	1	0
3	8	-5	1	1
3	9	-6	1	1
4	3	1	0	0
4	5	1	1	1
4	6	1	1	1
4	7	1	1	1
4	7	1	1	1
5	2	3	0	0
5	6	-1	1	1
5	8	-3	1	1
5	11	-6	1	1
6	4	2	0	0
6	8	-2	1	1
6	8	-2	1	1
6	9	-3	1	1
6	11	-5	1	1
6	12	-6	1	1
7	5	2	0	0
7	7	0	1	0
7	10	-3	1	1
7	10	-3	1	1
8	8	0	1	0
8	9	-1	1	1
8	11	-3	1	1
9	7	2	0	0
10	5	5	0	0
10	10	0	1	0
11	9	2	0	0
12	9	3	0	0

Classic Analytic Approaches

There are three common classic approaches to statistical analysis for designs in which there are matched pairs or grouped (blocked) data. The first approach is using the paired *t*-test which requires the usual assumptions of linear models—a sufficient sample size and normal distri-

bution of the data. Using a paired *t*-test we derived a mean difference of -1.267 between groups (95% CI: -2.36, -0.17), $p \leq 0.025$.

The second approach is a non-parametric Wilcoxon signed-rank test³² which tests equality of matched pairs of observations (the null hypothesis is that the distributions are the same). Using this test we obtain $p \leq 0.027$.

A third approach, an extension of the Wilcoxon signed-rank test, is the Hodges-Lehmann treatment effects estimator with distribution-free confidence intervals.³³ This method entails estimating the average difference in outcomes (*x-y*) for all $n(n+1)/2$ possible pairs and then deriving the overall median of all averages (the Hodges-Lehmann estimator). Using this approach, we derive a median difference estimate of -1.5 (95% CI: -2.5, 0).

All three approaches were computed using Stata (StataCorp. 2017. *Stata Statistical Software: Release 15*. College Station, TX: StataCorp LLC). The Hodges-Lehmann model was estimated using the user-written module ALIGNEDPAIRS.³⁴

ODA Fixed Discriminant Threshold

Hamilton scale scores (Table 1) were compared between Imipramine vs. Placebo patient groups via exploratory ODA using fixed discriminant threshold values.³⁵ Analysis identified a training model (if score ≤ 6 predict group=Imipramine, otherwise predict group=Placebo) which yielded marginally significant ($p < 0.090$) moderate effect strength (ESS=30.0).

A directional fixed-threshold ODA was also conducted to test the *a priori* confirmatory hypothesis that patients in the Imipramine group would have lower Hamilton depression scores than patients in the Placebo group. The same model (and associated ESS) identified in non-directional training analysis also emerged in confirmatory analysis, however the effect was statistically significant ($p < 0.043$). One-sample (leave-one-out, or LOO) cross-generalizability analysis revealed that while model sensitivity in

predicting Imipramine patients was unchanged in training and LOO analysis, LOO sensitivity for Placebo patients fell (from 70% in training) to 56.7%, yielding a relatively weak (ESS=16.7) statistically insignificant ($p < 0.16$) effect.

Thus, assessed via a fixed discriminant threshold applied to the entire sample, scores on the Hamilton Scale were unable to reproducibly discriminate Imipramine *vs.* Placebo patients. This is due to misclassification of Imipramine group patients having scores at or near (i.e., 5-7) the model threshold (6 points)—which is half of the maximum observed score (12 points, Table 1). Only *if* it is hypothesized that Imipramine patients had lower depression scores than Placebo patients, *and* if only training results are of interest, *then* the moderate training effect (ESS=30) was statistically significant ($p < 0.043$).

ODA Relative Discriminant Threshold

Two directional hypotheses and corresponding relative threshold criteria used to evaluate each pairwise comparison are: (1) the Hamilton scale score of the Imipramine patient is less than or equal to the Hamilton scale score of the Placebo patient; and (2) the Hamilton scale score of the Imipramine patient is strictly less than the Hamilton scale score of the Placebo patient.

As seen in Table 1, by the first criterion 22 of 30 Imipramine patients had a Hamilton scale score which was less than or equal to the corresponding score of the paired Placebo patient. If it is assumed that Hamilton scale score is a uniform random variable then $p(\text{success}) = 0.50$ for each pair, and the binomial probability of 22 successes in 30 trials is $p < 0.00545$. By the second criterion, 18 of 30 Imipramine patients had a Hamilton scale score which was strictly less than the corresponding score of the paired Placebo patient ($p < 0.0806$).

Comments

As is often the case, using alternative statistical methods produced different analytic conclu-

sions.³⁶ Considering legacy methods first, the parametric paired *t*-test found a statistically significant difference in mean depression scores between Imipramine *vs.* Placebo groups—but Table 1 indicates markedly skewed data for the former group thereby invalidating the assumed normality and calling into question the validity of the obtained *p*-value. While non-parametric Wilcoxon signed-rank test^{37,38} found depression scores of Imipramine patients had significantly lower ranks *vs.* matched Placebo patients, distribution-free confidence intervals for the Hodges-Lehmann treatment effects estimator indicated this effect overlapped zero—thereby indicating a non-significant difference.

Next consider the findings obtained via ODA. First, for a *fixed* discriminant threshold a marginal training effect emerged in *exploratory* analysis, and a significant training effect in *confirmatory* analysis—which became insignificant in *LOO* analysis due to misclassification of Imipramine patients with depression scores near the threshold. Thus, while the confirmatory model explains results obtained for the *present* sample, comparable predictive accuracy is *not* expected by applying the *identical* threshold to classify an *independent* random sample. Second, for a *relative* threshold, a statistically significant effect was obtained for the hypothesis that depression scores of Imipramine patients were less than or equal to (never greater than) depression scores of matched Placebo patients, and a marginally significant effect emerged if it was hypothesized that depression scores of Imipramine patients were strictly lower (always less) than depression scores of matched Placebo patients.

Which analytic finding should be used? The answer to this question depends upon the conceptual orientation of the researcher. If one justifies on a theoretical basis that *means* are the appropriate moment with which to compare the response distributions of the groups, then *t*-test should be reported (presently the validity of the *p*-value is suspect). If one's theoretical orientation indicates that *rankings* are the appropriate

moment to compare response distributions of the groups then the Wilcoxon signed-rank test using the Hodges-Lehmann treatment effects estimator should be reported (presently the confidence interval for the group comparison overlaps zero). And, if one's theoretical orientation is that the appropriate way to compare response distributions of groups is to compare the entire response distributions—identify the model that explicitly maximizes the effect strength normed *vs.* chance, obtain exact *p*-values while making no distributional assumptions, and estimate the cross-generalizability of the model—then ODA with fixed (for robust effects) or relative (for subtle, pairwise effects) should be reported.

The study data are a good pilot sample. Given failure of the training effect to reproduce in LOO analysis for the fixed threshold model, and failure of the “strictly-less-than” relative threshold model to reach statistical significance, it is natural to wonder how to improve the study and obtain a statistically significant, moderate (or stronger) effect strength in cross-generalizability analysis (ODA routinely employs LOO reproducibility analysis, which isn't available for the legacy methods). Achieving statistical significance is easily done (and thus is basically meaningless) by using a larger sample—the size is computed based on the LOO results obtained presently (the first axiom of novometric theory is the sample must provide adequate statistical power to test the alternative hypothesis^{39,40}). Increasing the model validity effect strength is more challenging (and thus theoretically and/or translationally meaningful), requiring improving the measurement precision of the attribute, particularly in the response scale region close to the value of the fixed threshold⁴¹, and/or replacing the singular depression score with a battery of measures offering greater theoretical clarity, and measurement granularity and precision.⁴²⁻⁴⁴

Another, arguably the most influential aspect of the analysis in need of consideration, is the method used in the matching process—or blocking, as Fleiss³¹ states. Given that the out-

come analysis may be biased due to confounding, it is imperative that the matching/blocking process eliminates confounding of observed covariates. In small samples with few covariates, matching directly on the available covariates may suffice. However, as the number of covariates increases, and as their distribution differs between the treatment and control conditions, methods which stratify and weight individuals into blocks of the propensity score to adjust for observed confounding should be considered.⁴⁵⁻⁴⁸

These techniques are incorporated within the ODA, CTA, and novometric frameworks. For example, in any given substantive area of scientific application, optimal (“maximum-accuracy”) methods are available to assist researchers to obtain a clear understanding of the factors which must be considered as being potential threats to causal inference: to identify variables characterizing participation in both discretionary treatment⁴⁹ and observational²⁶ research (i.e., to identify possible confounding variables), and to identify structural breaks in single^{50,51} and multiple-group⁵² interrupted time series analysis, dose-response studies^{53,54} and in research investigating mediating processes.⁵⁵ Optimal ODA and CTA methods are available to assist researchers to identify and correct (in real time) covariate interactions which exist in data from matched⁴⁶ and randomized trials⁵⁶ to remove otherwise undetected threats to causal inference. Globally-optimal (i.e., novometric) analysis is available to identify all statistically unique propensity score models that maximize classification accuracy and vary as a function of complexity, which exist within a sample: this makes model misspecification impossible, and is used in both time-to-event and single-case precision forecasting.⁵⁷⁻⁶⁰

The present findings further illustrate why we strongly advocate using the ODA, CTA and novometric frameworks to draw causal inferences about treatment effects in observational data and in data from randomized controlled trials. Clearly, changes are needed in guidelines

concerning how health care interventions and policy changes are evaluated.^{61,62}

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Author Notes

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