

Dealing with "some" important statistical assumptions.

1) The issue of normality (today):

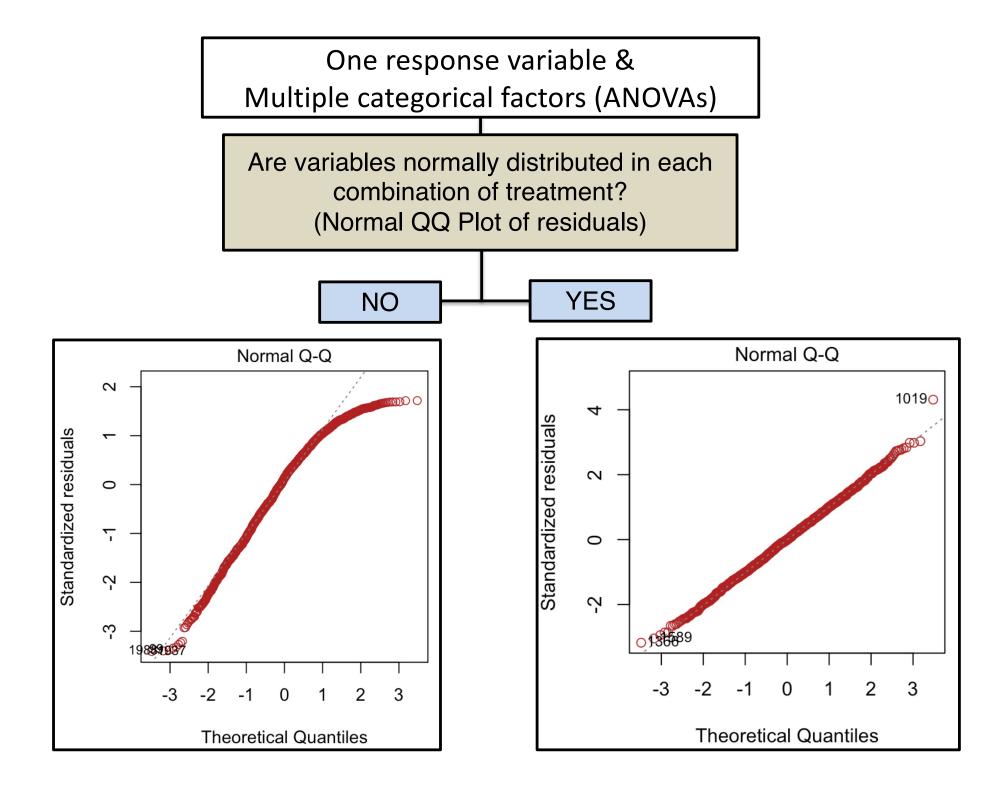
- Parametric (e.g., ANOVA): assume parametrized families of probability distributions (e.g., normal defined by two parameters, i.e., mean and variance). Parameter estimates tend to be sensitive to nonnormality (e.g., issue in regression slopes), but not necessarily in statistical hypothesis testing (P-values may be not as sensitive).

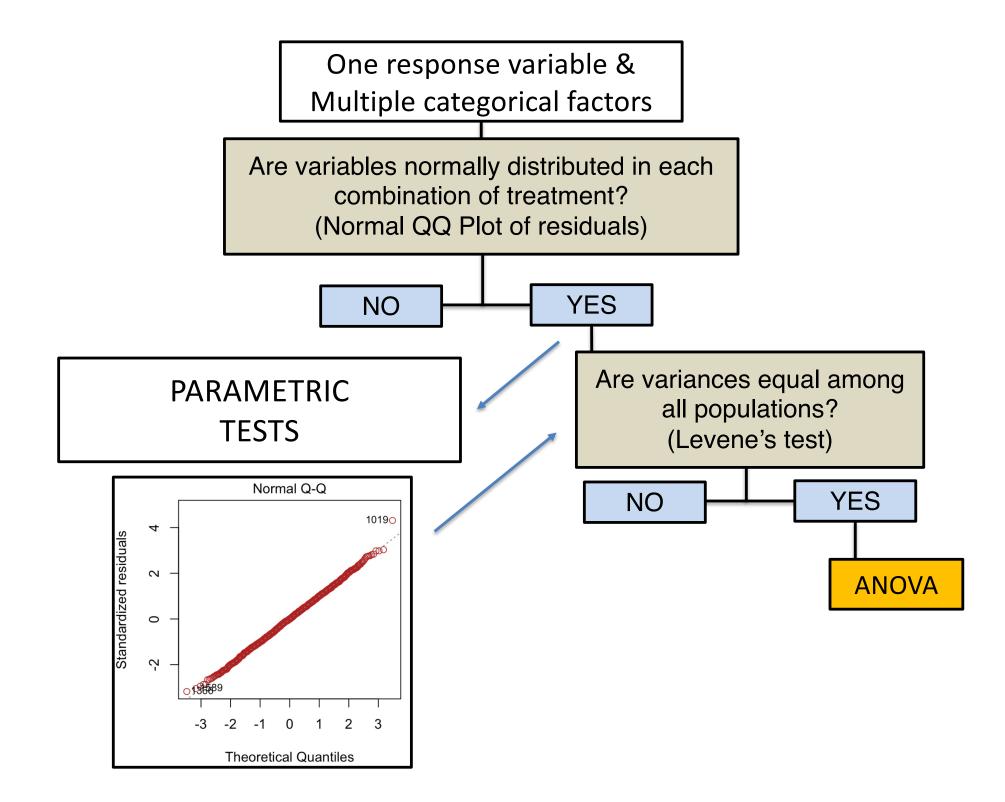
- Non-parametric: either distribution free (e.g., permutation tests) or ranked based tests.

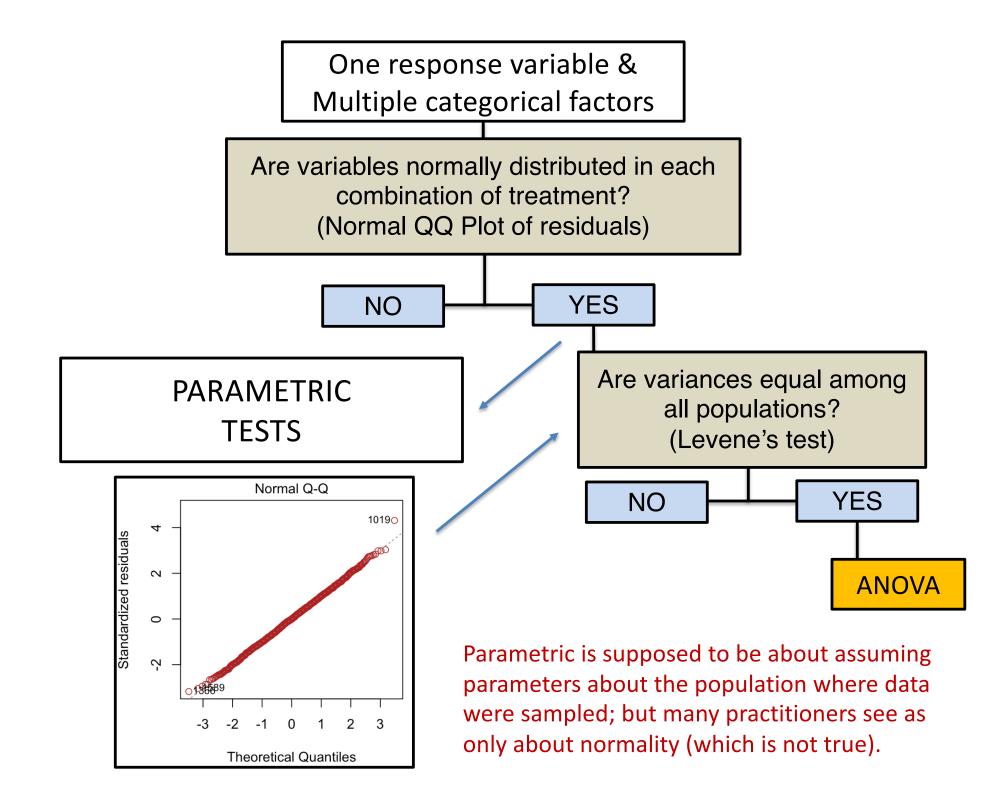
Dealing with "some" important statistical assumptions.

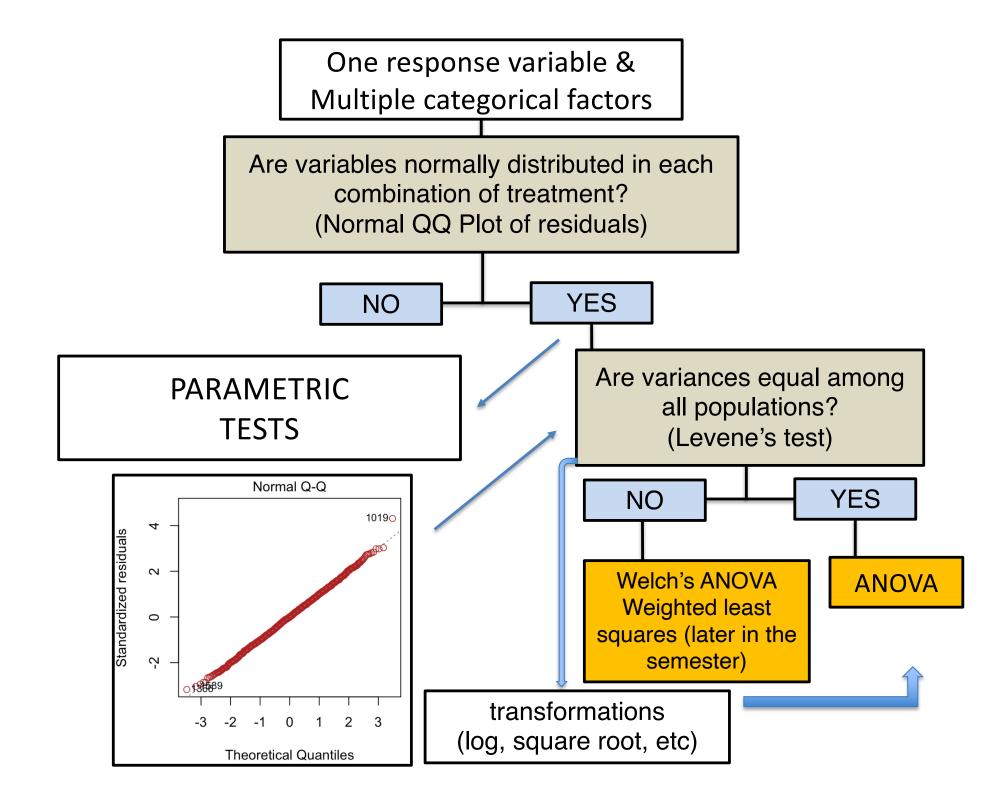
2) The issue of homogeneity of variances (later in the course):

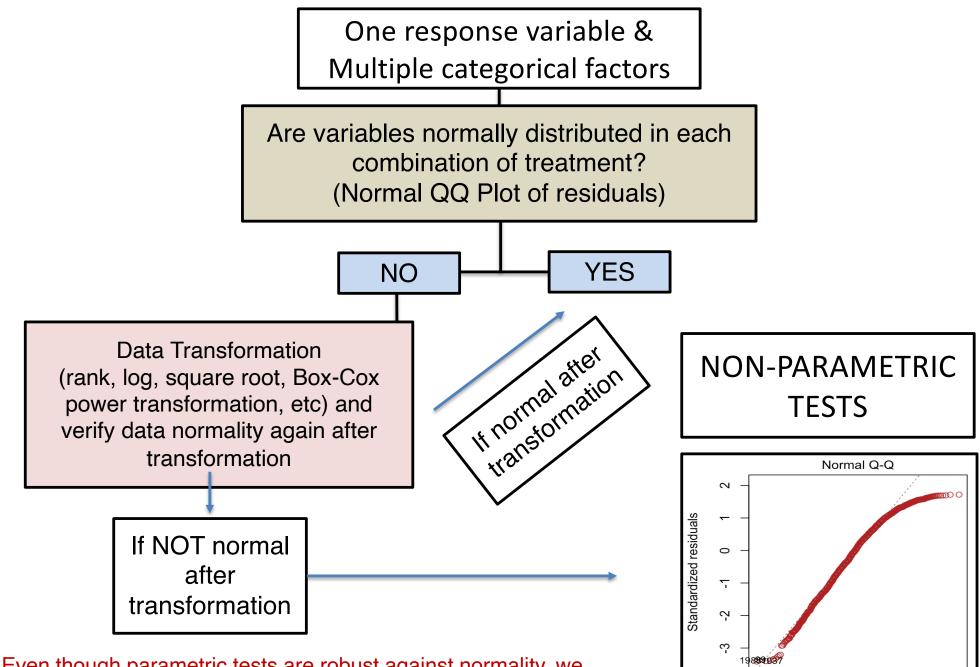
- Standard (e.g., ANOVAs, regressions) assume homoscedasticity.
- Robust approaches (Welch's ANOVA, Weighted least squares) are good to deal with heteroscedasticity.











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-1

0

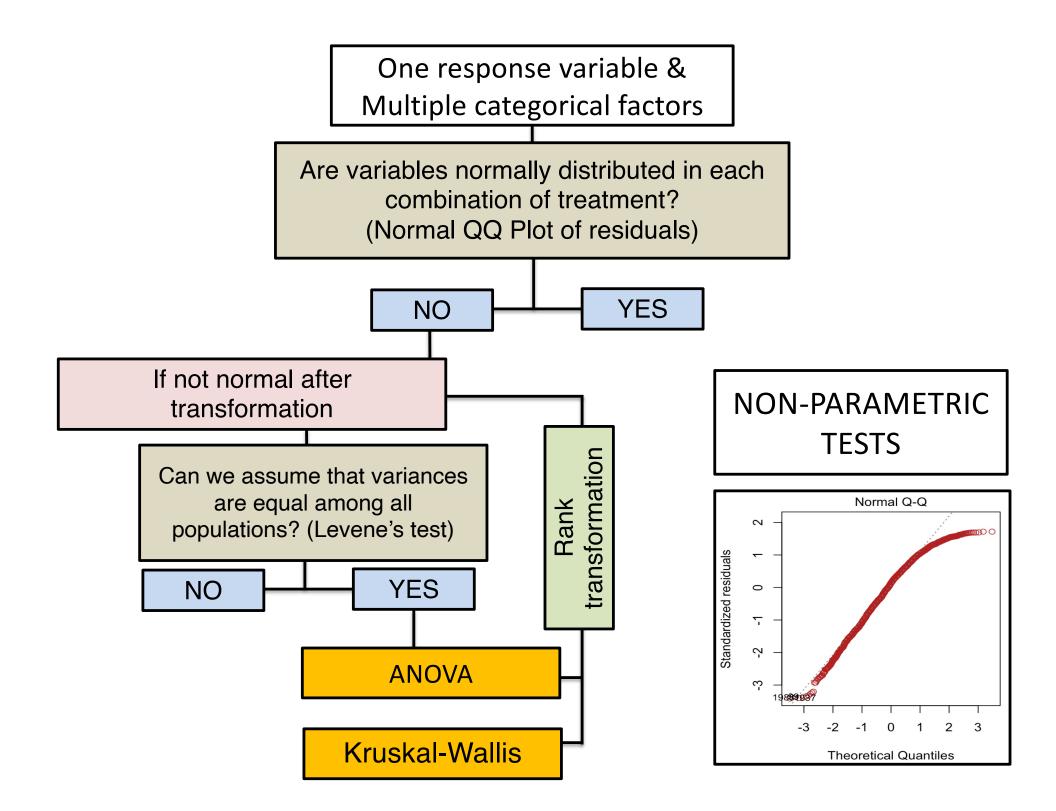
Theoretical Quantiles

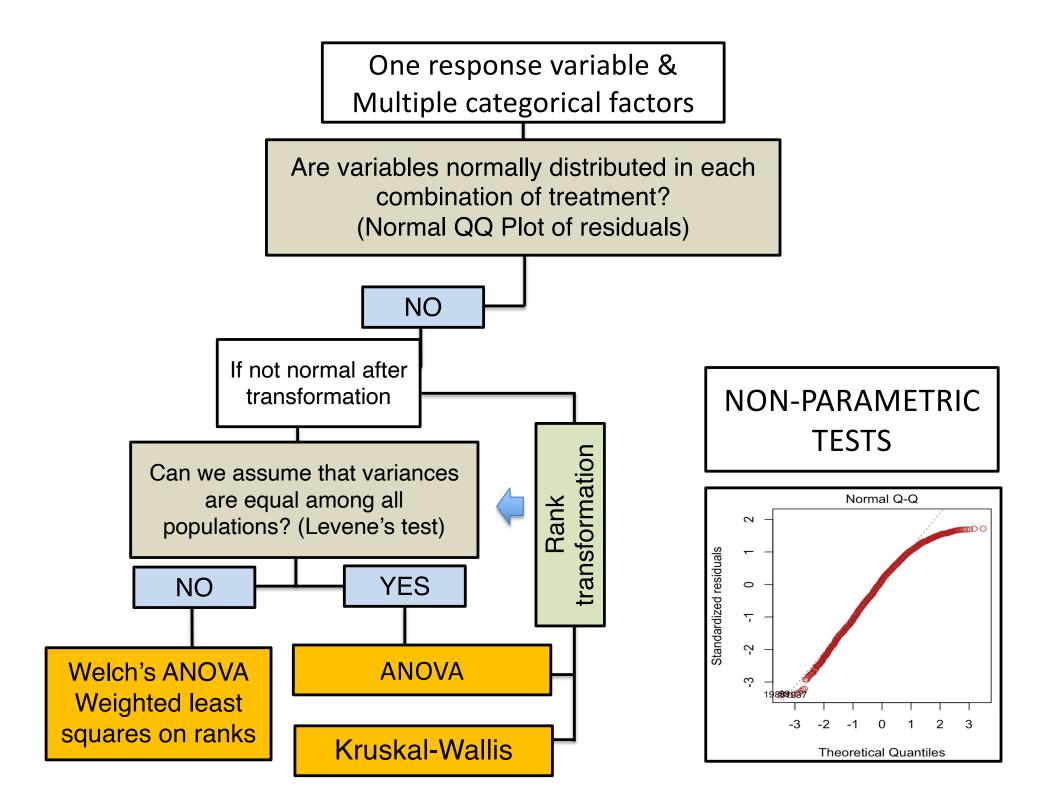
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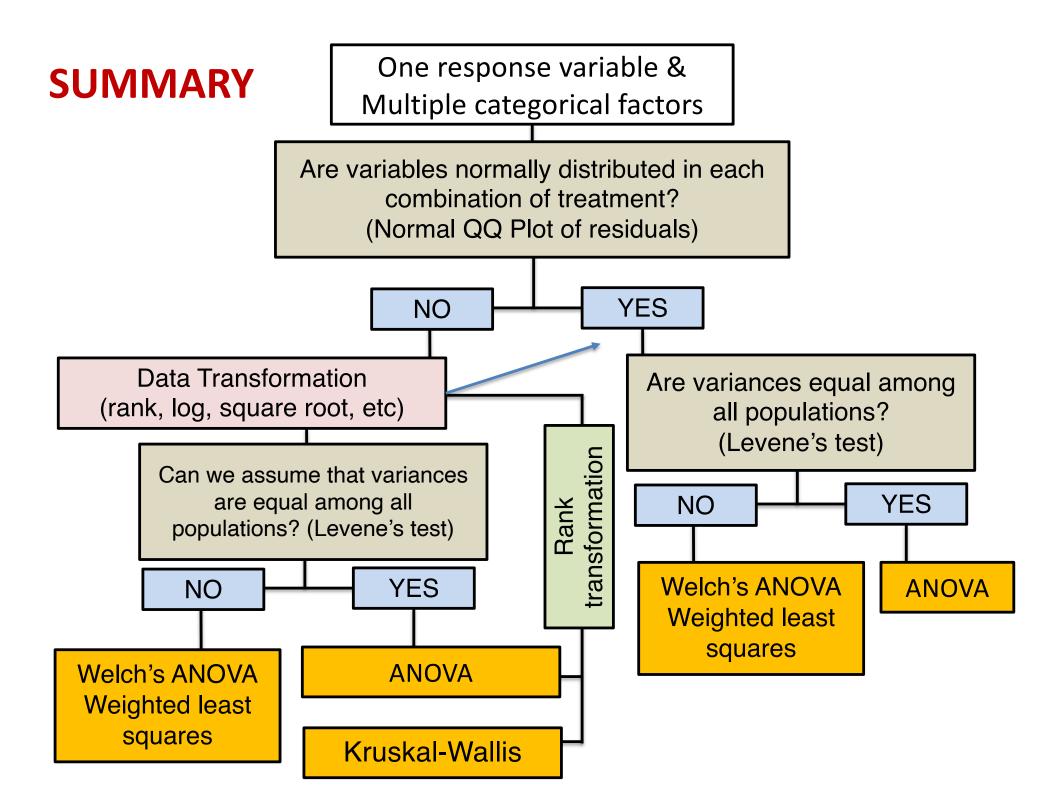
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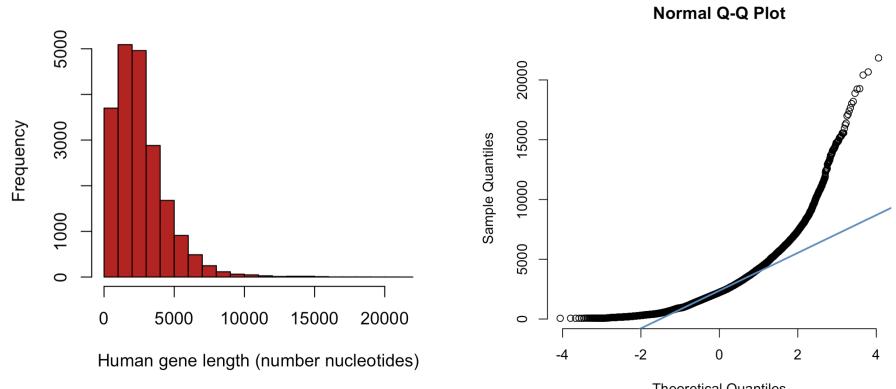
Even though parametric tests are robust against normality, we often don't know how much for the particular data at hands; the tradition is then to use non-parametric tests







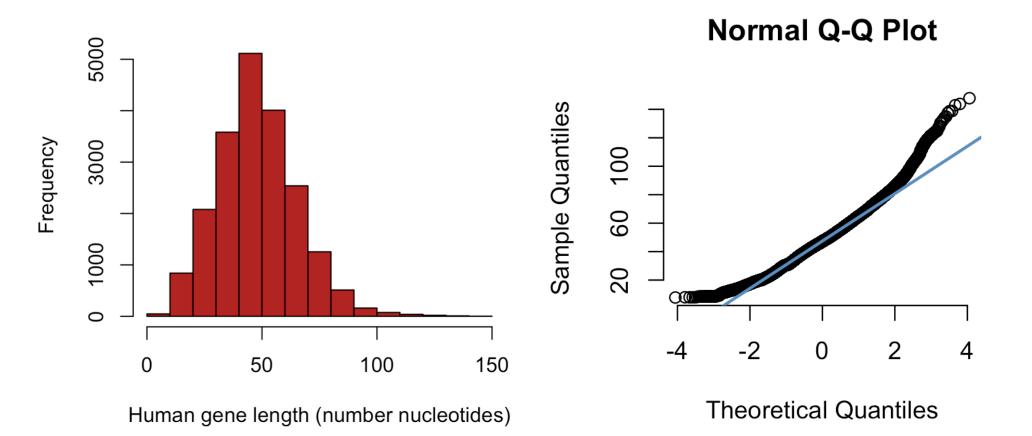
improve normality (today) & homoscedasticity (covered in another lecture)



Theoretical Quantiles

improve normality & homoscedasticity (another lecture)

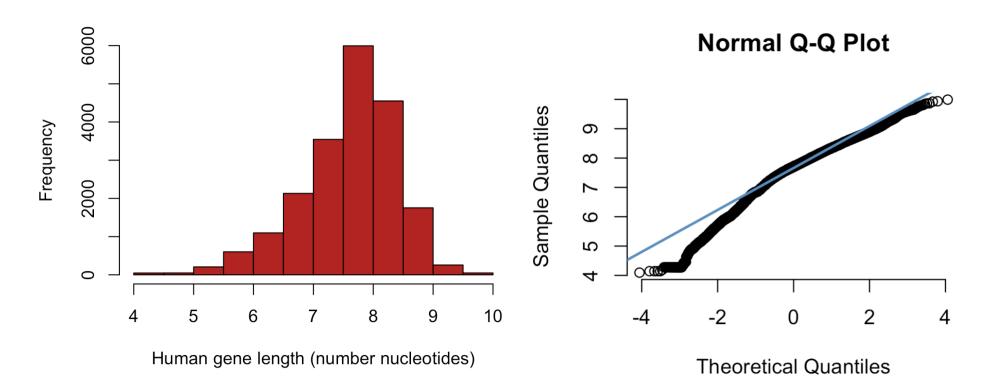
square-root transformation



The role of data transformations

improve normality & homoscedasticity (another lecture)

log transformation



A few words on data transformation

One size may not fit all:

1) One transformation may help approximate normality, but another transformation may be required to approximate homoscedasticity (e.g., log(sqrt(data))).

2) One transformation may negate (reverse) the other – the one that makes the data approximate homoscedasticity may make data non-longer normal.

3) If data are complex (e.g., several predictors in a regression model), it may not be possible that one single transformation will allow data to behave properly under assumptions.

Possible solution: focus on analytical solutions (many covered in this course) and not always transformations; or combine different transformation.

A few words on data transformation

3) If data are complex (e.g., several predictors in a regression model), it may not be possible that one single transformation will allow data to behave properly under assumptions.

Possible solution: focus on analytical solutions (many covered later in the semester) and not always transformations; or combine different transformation.

The R Package trafo for Transforming Linear Regression Models

Lily Medina Humboldt Universität zu Berlin Piedad Castro Humboldt Universität zu Berlin

Ann-Kristin Kreutzmann Freie Universität Berlin Natalia Rojas-Perilla Freie Universität Berlin

Abstract

The linear regression model has been widely used for descriptive, predictive, and inferential purposes. This model relies on a set of assumptions, which are not always fulfilled when working with empirical data. In this case, one solution could be the use of more complex regression methods that do not strictly rely in the same assumptions. However, in order to improve the validity of model assumptions, transformations are a simpler approach and enable the user to keep using the well-known linear regression model. But how can a user find a suitable transformation? The R package **trafo** offers a simple userfriendly framework for selecting a suitable transformation depending on the user needs. The collection of selected transformations and estimation methods in the package **trafo** complement and enlarge the methods that are existing in R so far.



Pedro Peres-Neto, PhD @com_ecology

Most often, the more important question is how lack of normality affects estimates & inference; for that, we can make such assessments using simulations under the model of interest.

🚳 Mason Fidino, PhD @masonfidino · Jan 27

Reviewing a paper that uses a shapiro-wilk test to see if their response variable is normally distributed before using linear regression. This is not necessary! Linear regression does not assume a normally distributed response, it's the residuals that are normally distributed.

Show this thread

```
1 $et.seed(3)
3 n <- 500
4 # create covariate
5 covariate <- runif(n, -10, 10)
6
7 # generate response variable
8 y <- rnorm(n, 1 + 2 * covariate, 5)
9
10 # oh no, not normally distributed (p < 0.05)!
11 shapiro.test(y)
12 # Shapiro-Wilk normality test
13 #
14 # data: y
15 # W = 0.98609, p-value = 0.0001039
16
17 # fit linear regession anyways
18 ml <- lm(y ~ covariate)
19
20 # get model residuals, this is what we assume to
21 # normally distributed.
22 m_resid <- resid(m1)
23
24 # oh wow, normally distributed (p > 0.05)!
25 shapiro.test(m_resid)
26 # Shapiro-Wilk normality test
27 #
28 # data: m_resid
29 ** data: m_resid
20 ** get m_resid
20 ** get m_resid
21 ** on m_resid
22 ** data: m_resid
23 ** data: m_resid
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```

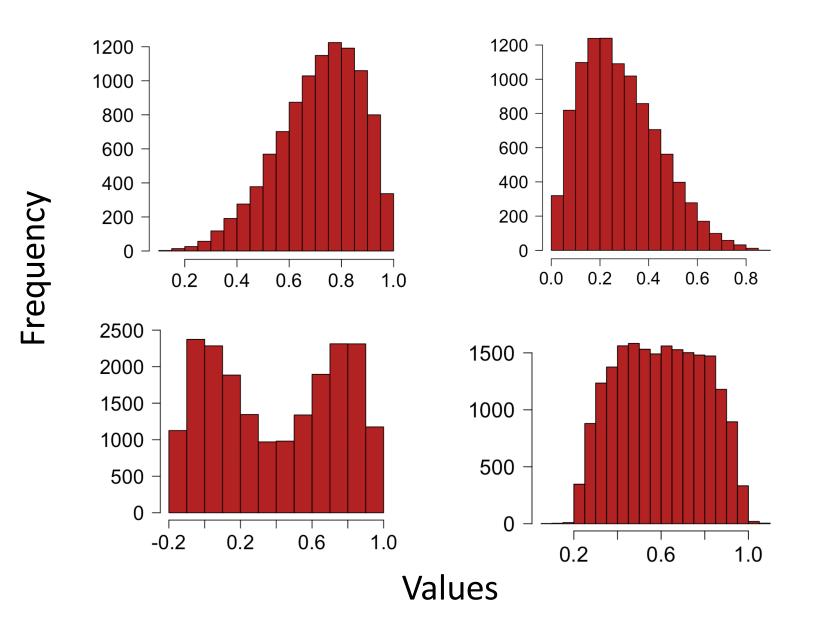
7:41 PM · Jan 30, 2023 · 492 Views

Assumptions in social media

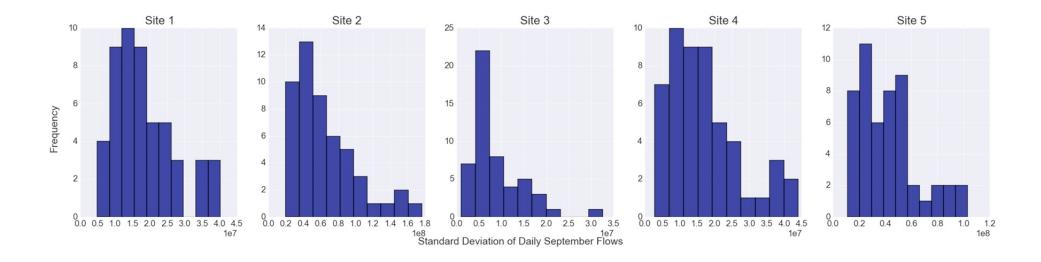
The effects of non-normality on statistical inference



Dealing with non-normality in statistical inference hypothesis testing



Dealing with non-normality in statistical inference – hypothesis testing



Non-normal distributions have many shapes and would be quite hard to develop sampling distributions for all these different shapes

(though it can and has been done in more advanced analysis)

Parametric tests assuming normality (e.g., t-test & ANOVA) are often robust against non-normality; but depending on the type of non-normality (shape), parametric tests can have type I errors different (often greater) from alpha; and low power (increased type II error).

One challenge is to separate normality from heteroscedasticity issues (even in simulations).

The other challenge is when samples come all from populations with different distributions (even though they could have the same means, i.e., H_0 is true).

Parametric tests assuming normality (e.g., t-test & ANOVA) are often robust against non-normality; but depending on the type of nonnormality (shape of the distribution), parametric tests can have type I errors (false positives) that differ (often greater) from alpha; and low power (increased type II error; false negatives).

Br J Math Stat Psychol. 2013 May;66(2):224-44. doi: 10.1111/j.2044-8317.2012.02047.x. Epub 2012 May 24.

The impact of sample non-normality on ANOVA and alternative methods.

Lantz B¹.

Author information

Abstract

In this journal, Zimmerman (2004, 2011) has discussed preliminary tests that researchers often use to choose an appropriate method for comparing locations when the assumption of normality is doubtful. The conceptual problem with this approach is that such a two-stage process makes both the power and the significance of the entire procedure uncertain, as type I and type II errors are possible at both stages. A type I error at the first stage, for example, will obviously increase the probability of a type II error at the second stage. Based on the idea of Schmider et al. (2010), which proposes that simulated sets of sample data be ranked with respect to their degree of normality, this paper investigates the relationship between population non-normality and sample non-normality with respect to the performance of the ANOVA, Brown-Forsythe test, Welch test, and Kruskal-Wallis test when used with different distributions, sample sizes, and effect sizes. The overall conclusion is that the Kruskal-Wallis test is considerably less sensitive to the degree of sample normality when populations are distinctly non-normal and should therefore be the primary tool used to compare locations when it is known that populations are not at least approximately normal.

Parametric tests assuming normality (e.g., t-test & ANOVA) are often robust against non-normality; but depending on the type of non-normality (shape), parametric tests can have type I errors different (often greater) from alpha and also low power (increased type II error).

What happens if the Type I error probability (rate) is *greater* than alpha? i.e., increase number of False Positives.

Parametric tests assuming normality (e.g., t-test & ANOVA) are often robust against non-normality; but depending on the type of non-normality (shape), parametric tests can have type I errors different (often greater) from alpha and also low power (increased type II error).

What happens if the Type I error probability (rate) is *greater* than alpha? i.e., increase number of False Positives.

What happens if the Type I error probability (rate) is *smaller* than alpha? decrease False Positives but also decrease True Positives (i.e., lower statistical power).

Type I versus Type II errors – the "common" view

A **Type** I **error (false positive)** is an **error** in every sense of the word. A conclusion is drawn that the null hypothesis is false when, in fact, it is true.

Therefore, **Type I** errors are generally considered more serious than **Type II** errors (false negatives).

Type II errors are often considered as "oh well, we were not able to detect an effect"...perhaps increase sample size!

Adapted from http://davidmlane.com/hyperstat/A2917.html

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Adapted from http://davidmlane.com/hyperstat/A2917.html

When committing a type I error, you are stating that something that is false to be true.

CONFUSING: When committing a type II error, you are NOT stating that something that is true to be false (you are just not discovering something new).

Non-parametric tests based on ranks are those that can handle non-normal data

These are the main tests traditionally used in Biology for comparing samples:

1) For comparing two samples (analogue of the parametric two sample t-test) – *The Mann–Whitney U-test* (also known as the Mann–Whitney–Wilcoxon test, the Wilcoxon rank-sum test, or the Wilcoxon two-sample test).

Non-parametric tests based on ranks are those that can handle non-normal data

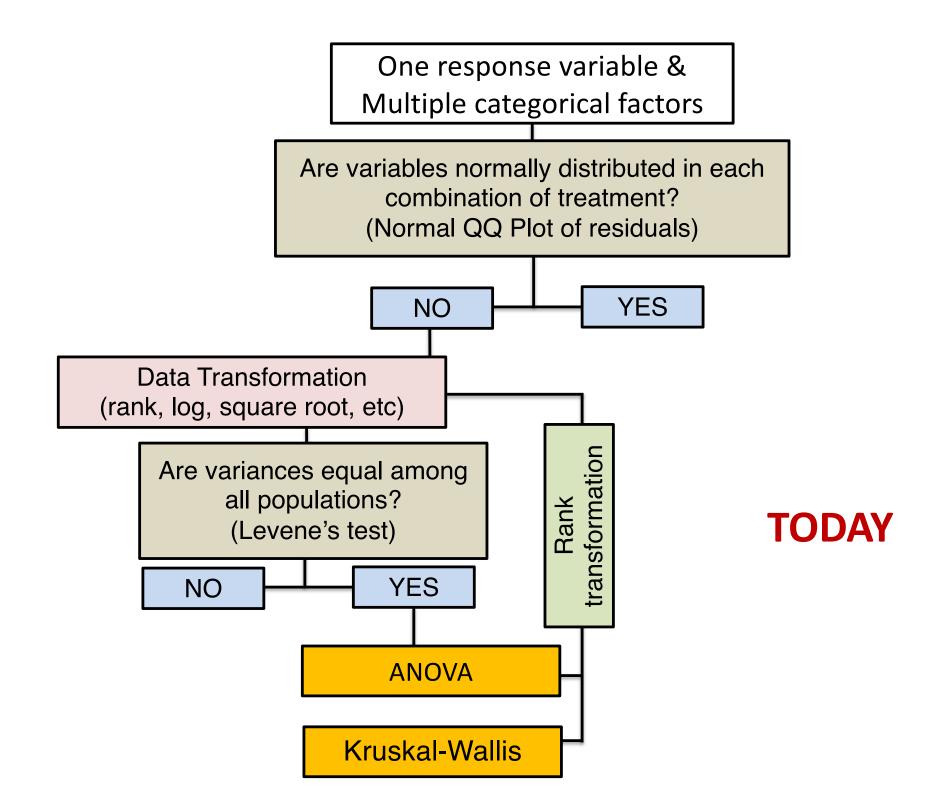
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2) For comparing multiple samples (analogue of the parametric ANOVA) – *The Kruskal-Wallis test* (generalization of the U-test)

The P-value for the *The Mann–Whitney U-test and the The Kruskal-Wallis test* is mathematically the same; as such, we will cover only the latter.

Note: remember that $t^2 = F$; we often cover t-tests (and not only ANOVAs) in courses for two main reasons – [1] one sample t-tests; [2] understand the nature of post-hoc testing (e.g., post-hoc pairwise comparisons of means after ANOVA and because there is a t-test dealing with samples when their populations differ in their variances).



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gene	class	F _{ST}
CVJ5	DNA	-0.006
CVB1	DNA	-0.005
6Pgd	protein	-0.005
Pgi	protein	-0.002
CVL3	DNA	0.003
Est-3	protein	0.004
Lap-2	protein	0.006
Pgm-1	protein	0.015
Aat-2	protein	0.016
Adk-1	protein	0.016
Sdh	protein	0.024
Acp-3	protein	0.041
Pgm-2	protein	0.044
Lap-1	protein	0.049
CVL1	DNA	0.053
Mpi-2	protein	0.058
Ap-1	protein	0.066
CVJ6	DNA	0.095
CVB2m	DNA	0.116
Est-1	protein	0.163

Example: F_{ST} is a measure of the amount of geographic variation in a genetic polymorphism. Here, McDonald et al. (1996) compared two populations of the American oyster regarding the F_{ST} based on six anonymous DNA polymorphisms (variation in random bits of DNA of no known function) and compared them to F_{ST} values on 13 proteins.

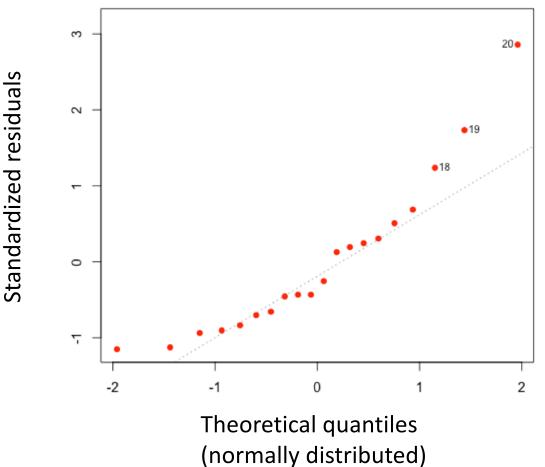
Question: Do protein differ in F_{ST} values in contrast to anonymous DNA polymorphisms?

Zero F_{ST} = no genetic variation (panmictic) **negative** F_{ST} = more genetic variation within populations than between the two populations being compared.

positive F_{ST} = more variation between populations than within the two populations being compared.

e sum of the ranks for each group, then the test statistic, H. H is given by a primula that the form of the state of the second se

F_{st} data highly non-normal, so transformation is advised; let's apply the rank transformation



Normal Q-Q normal residual plot for the t-test

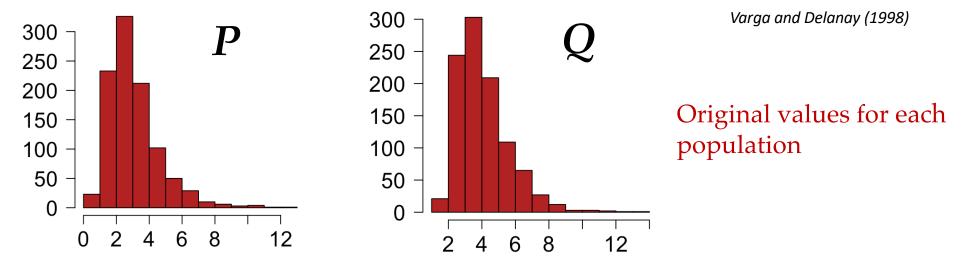
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gene	class	F _{ST}	R	ank	Rank	
CVJ5	DNA	-0.006	1	1		
CVB1	DNA	-0.005		2.5		(2+3)/2=2.5
6Pgd	protein	-0.005			2.5	(2+3)/2-2.3
Pgi	protein	-0.002			4	
CVL3	DNA	0.003		5		•
Est-3	protein	0.004			6	•
Lap-2	protein	0.006			7	•
Pgm-1	protein	0.015			8	•
Aat-2	protein	0.016	[9.5	(0, 10)/2 0 E
Adk-1	protein	0.016			9.5	(9+10)/2=9.5
Sdh	protein	0.024			11	
Acp-3	protein	0.041			12	•
Pgm-2	protein	0.044			13	•
Lap-1	protein	0.049			14	•
CVL1	DNA	0.053		15		•
Mpi-2	protein	0.058			16	•
Ap-1	protein	0.066			17	
CVJ6	DNA	0.095		18		
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http://www.biostathandbook.com/kruskalwallis.html Data from McDonald et al. (1996) he sum of the ranks for each group, then the test statistic, H. H is given by a formula that basically represents the variance of the ranks among groups,

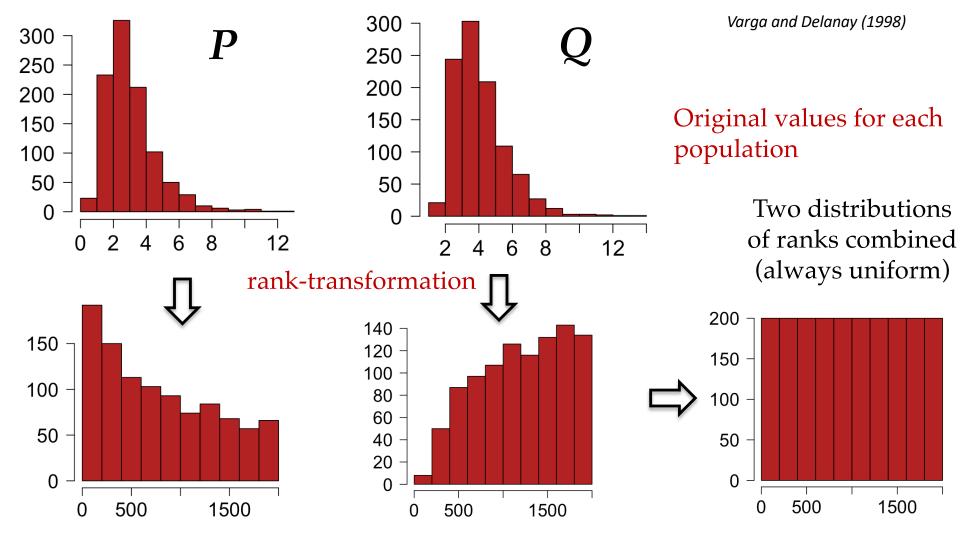
We want to know whether samples come from statistical populations that vary in their ranks

What is the probability that a randomly sampled observation from population P is greater (or smaller) in rank than a randomly sampled observation from Q? *If the probability is small, then the samples come from different populations!*



We want to know whether samples come from statistical populations that vary in their ranks – example from two large samples

What is the probability that a randomly sampled observation from population P is greater (or smaller) in rank than a randomly sampled observation from Q? *If the probability is small, then the samples come from different populations!*



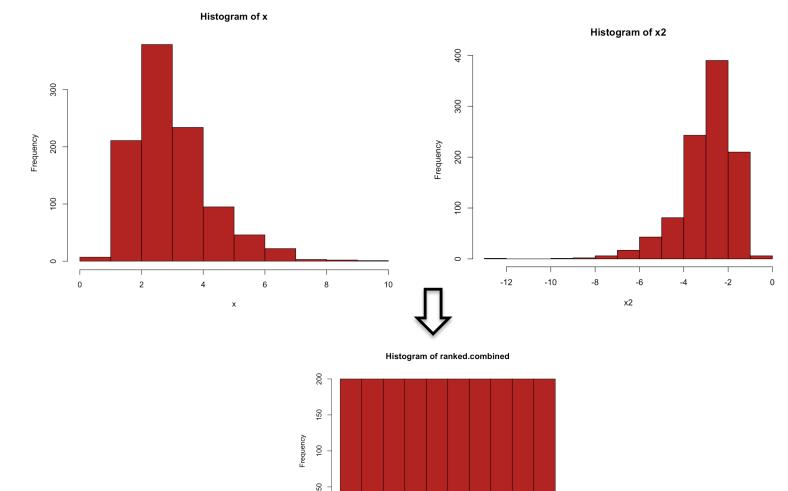
•••

x <- rlnorm(1000,1,0.4) hist(x,col="friebrick") x2 <- -rlnorm(1000,1,0.4) hist(x2,col="friebrick")

ranked.combined <- rank(c(x,x2))
hist(ranked.combined,col="friebrick")</pre>

Two distributions of ranks combined (always uniform)

Let's see that "manually" using R code



1000

1500

2000

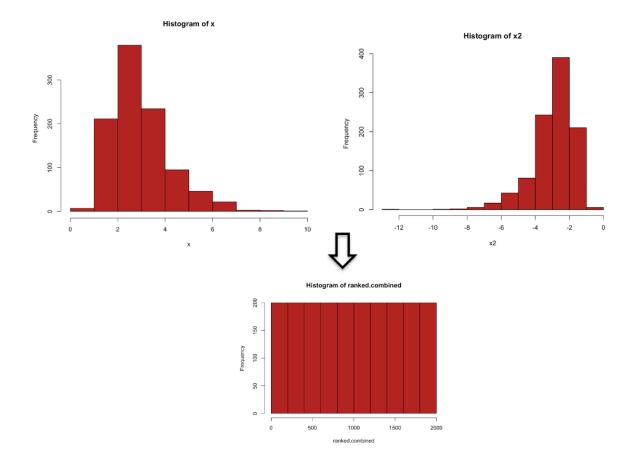
500

0

0

Ranked-based statistical tests remove the natural ways we think about the original units of the variables of interest

and they also reduce statistical power to detect true differences, i.e., increase type II error (false negatives).



Rank based tests

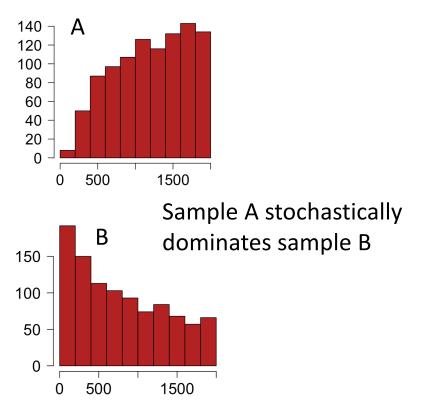


Kruskal-Wallis test (akin to one-factorial ANOVA but based on ranks)

Ho: no population from where the samples were taken stochastically dominates another population (stochastic homogeneity).

Ha: at least one population from where the sample was taken stochastically dominates another population (stochastic heterogeneity).

Used on ranks)



Kruskal-Wallis test (akin to one-factorial ANOVA but based on ranks)

H₀: no population from where the samples were taken stochastically dominates another population (stochastic homogeneity).

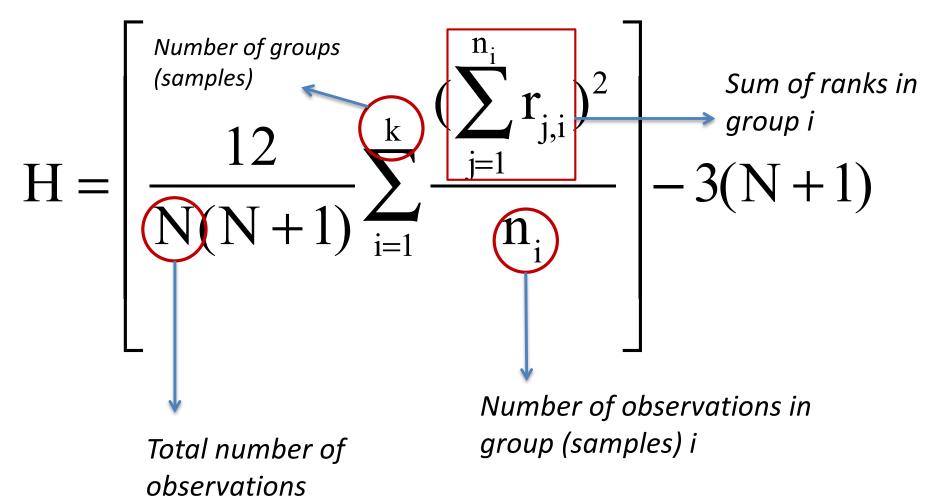
H_A**:** at least one population from where the sample was taken stochastically dominates another population (stochastic heterogeneity).

F_{STs} data –

 H_0 : DNA and protein do not stochastically dominate each other in their F_{STs} .

 H_A : Either DNA or protein stochastically dominate each other in their F_{STs} .

Kruskal-Wallis test – statistic H

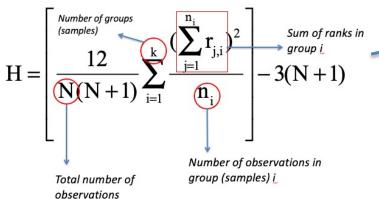


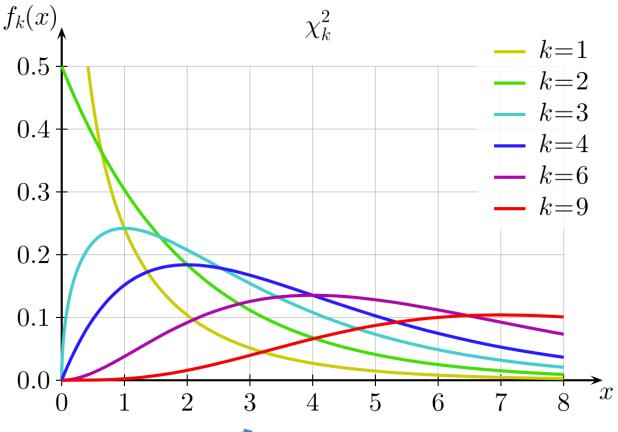
No need to memorize or understand this formula (F much more important) – but I think is relevant to understand that statisticians spend serious time on these formulae (or formulas).

Kruskal-Wallis test – statistic H

No need to memorize or understand this formula (keep your "energy" for F if you want to).

But I think is relevant to understand that statisticians spend serious time on those.





Equations also demonstrate the work others do to make test statistics (H here) to be contrastable to existing probability distributions (chi-square in this case) populations is highly skewed, so they analyzed the data with a Kruskal–Wallis

ting with a measurement variable, the Kruskal–Wallis test starts by substituting overall data set for each measurement value. The smallest value gets a rank of 1, allest gets a rank of 2, etc. Tied observations get average acts in the value of 1 the value of 2, etc. Tied observations get average acts in the value of 1 the value of 1 of 2, etc. Tied observations get average acts in the value of 1 of 2, etc. Tied observations get average acts in the value of 1, of 2, etc. Tied observations get average acts in the value of 1 of 2, etc. Tied observations get a rank of 2.5.

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$$H = \left[\frac{12}{20(20+1)} * \sum_{i=1}^{2} \frac{(\sum_{j=1}^{n_i} r_{j,i})^2}{n_i}\right] - 3(20+1)$$
$$H = \left[\frac{12}{20(20+1)} * (\frac{60.5^2}{6} + \frac{149.5^2}{14})\right] - 3(20+1)$$

$$H = [0.029 * (610.04 + 1596.45)] - 63 =$$

H = 0.0425
given by a

te the sum of the ranks for each **Sutp**, th**60**:5 tes**149:5**c, H. H is given by a ble formula that basically represents the variance of the ranks among groups, nent for the number of ties. H is approximately chi-square distributed, meaning bility of getting a particular value of H by chance, if the null hypothesis is true, is responding to a chi-square equal to H; the degrees of freedom is the number of 1. For the example data, the mean rank for DNA is 10.08 and the mean rank for B, H=0.043, there is 1 degree of freedom, and the *P* value is 0.84. The null t the F_{ST} of DNA and protein polymorphisms have the same mean ranks is not

ns given above, I think it would actually be better to analyze the oyster data with one-way P value of 0.75, which fortunately would not change the conclusions of McDonald et al.

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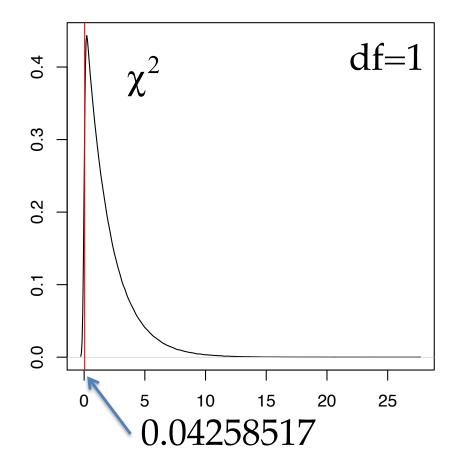
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Kruskal-Wallis test – statistic H

$$H_c = H / C_H = 0.0425 / 0.998 = 0.04258517$$

For small samples sizes (n <= 5), a special H distribution needs to be used (though R does not have it and uses the standard X²); if n > 5, then H follows a chi-square distribution with (k-1) degrees of freedom (df=2-1=1)



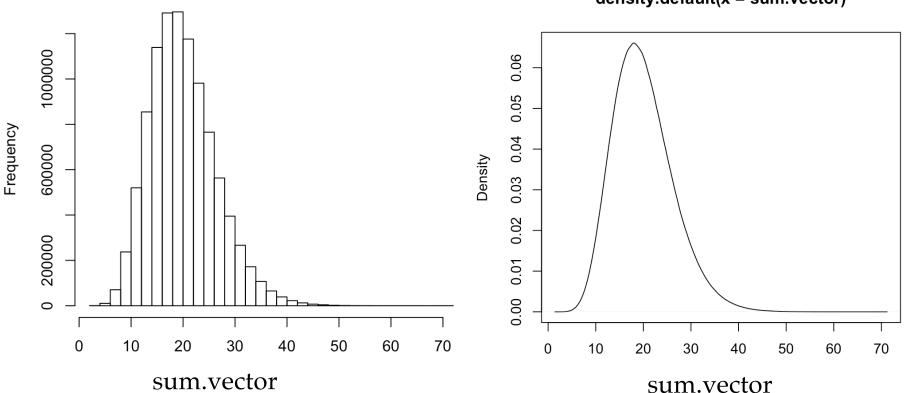
P=0.8365;

probability of finding by chance an H_c greater than the observed when assuming that H_0 is true. **Fun fact:** The chi-square distribution is the distribution of the sum of squared standard normal deviates.

Good place to generate more intuition about statistical distributions!

R code to generate the chi-square computationally *versus* analytically for 20 degree of freedom

The chi-square distribution is the distribution of the sum of squared standard normal deviates.

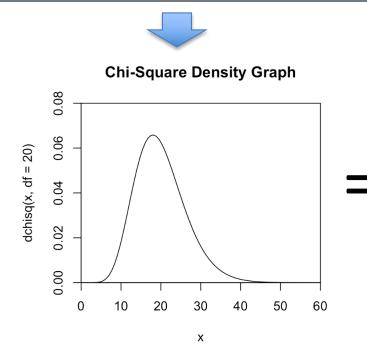


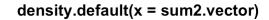
density.default(x = sum.vector)

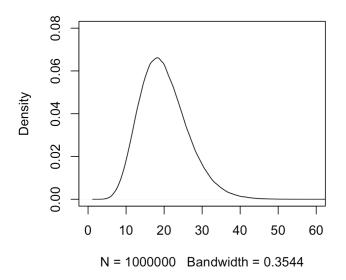
🗢 🔍 🔍 computational approach

samples <- replicate(1000000, rnorm(n=20))
sum2.vector <- apply(samples^2,2,sum)
plot(density(sum2.vector),xlim=c(0,60),ylim=c(0,0.08))</pre>

analytical approach







The chi-square distribution is the distribution of the sum of squared standard normal deviates.

fun fact: The F distribution is the ratio of two (scaled) chi-square distributed values. The scaling is done by appropriate division of degrees of freedom.

A general solution to rankbased tests



Kruskal-Wallis test is equivalent (close enough) to an ANOVA on ranks

Ho: no sample stochastically dominates another sample (stochastic homogeneity).

Ha: at least one sample stochastically dominates one other sample (stochastic heterogeneity).

"Stochastic homogeneity is equivalent to the equality of the expected values of the rank sample means. This finding implies that the null hypothesis of stochastic homogeneity can be tested by an ANOVA performed on the rank transforms, which is essentially equivalent to doing a Kruskal-Wallis H test."

Varga and Delanay (1998)

Journal of Educational and Behavioral Statistics Summer 1998, Vol. 23, No. 2, pp. 170–192

> The Kruskal-Wallis Test and Stochastic Homogeneity

> > András Vargha Eötvös Loránd University

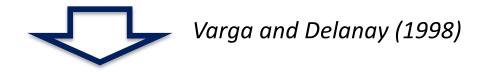
> > Harold D. Delaney University of New Mexico

Kruskal-Wallis test = ANOVA on ranks

Kruskal-Wallis:

Ho: no sample stochastically dominates another sample (stochastic homogeneity).

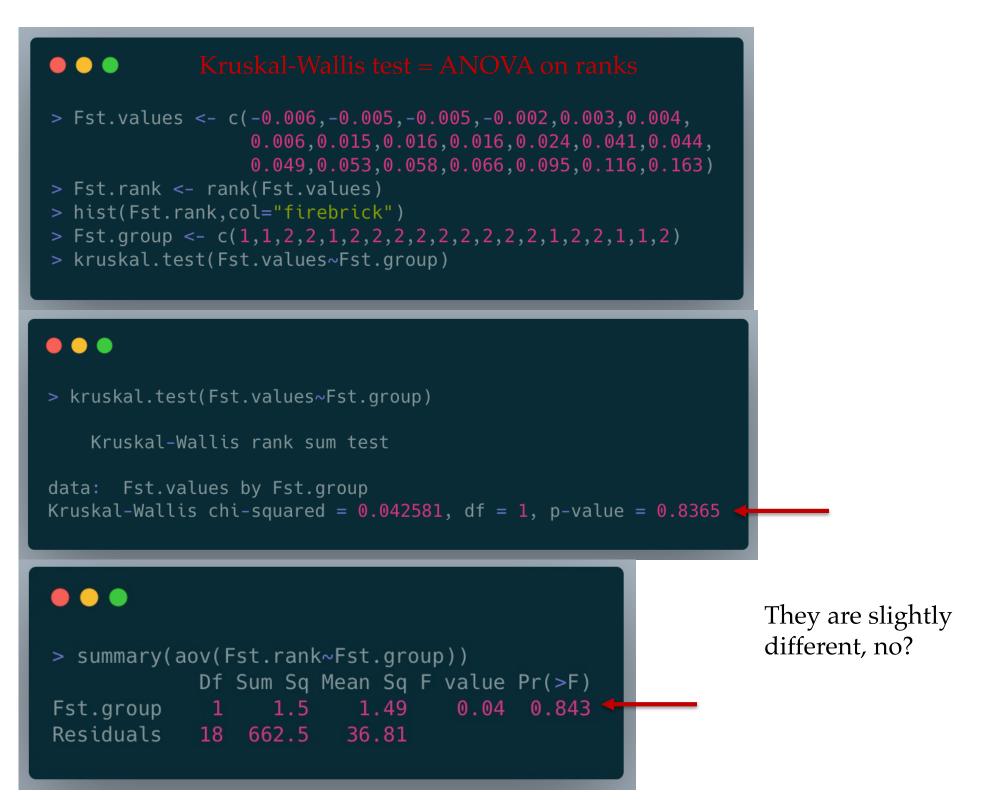
Ha: at least one sample stochastically dominates one other sample (stochastic heterogeneity).



ANOVA:

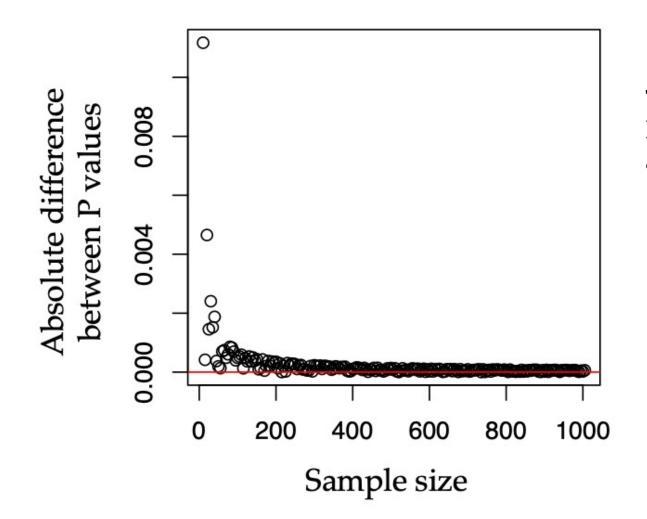
Ho: no mean differences in ranked values

Ha: at least one sample differs in mean ranked values from another sample



Kruskal-Wallis test = ANOVA on ranks

Kruskal-Wallis and ANOVA are "asymptotically equivalent" (i.e., the two functions "eventually" become "essentially **equal**") and so P-values are exactly the same for very large samples and they do not differ by much for small sample size.



Two sample Kruskal-Wallis P-values (chi-square based) and F-based P values) Kruskal-Wallis and ANOVA are "asymptotically equivalent" and so P-values are the same for very large samples and they do not differ by much for small sample size. Using R code to demonstrate the asymptotic equivalence.

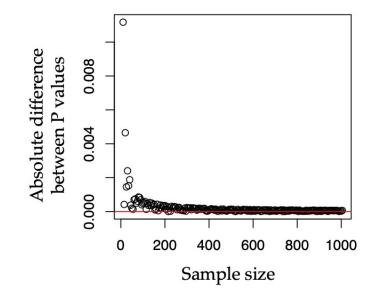
```
n.simul <- 200
Pvector <- matrix(0,n.simul,2)</pre>
n <- 10
n.vector <- matrix(0, n. simul, 1)
for (i in 1:n.simul){
  groups <- c(rep(1,n),rep(2,n))
  x \leftarrow rnorm(n*2)
  Pvector[i,1] <- kruskal.test(x~groups)$p.value</pre>
  Pvector[i,2] <- anova(lm(rank(x)~groups))$'Pr(>F)'[1]
  n < -n + 10
  n.vector[i] <- n
}
plot(n.vector/2,abs(Pvector[,1]-Pvector[,2]))
```

abline(h=0,col="red")

Kruskal-Wallis and ANOVA are "asymptotically equivalent"

•••

```
n.simul <- 200
Pvector <- matrix(0,n.simul,2)
n <- 10
n.vector <- matrix(0,n.simul,1)
for (i in 1:n.simul){
    groups <- c(rep(1,n),rep(2,n))
    x <- rnorm(n*2)
    Pvector[i,1] <- kruskal.test(x~groups)$p.value
    Pvector[i,2] <- anova(lm(rank(x)~groups))$'Pr(>F)'[1]
    n <- n+10
    n.vector[i] <- n
}
plot(n.vector/2,abs(Pvector[,1]-Pvector[,2]))
abline(h=0,col="red")</pre>
```



Kruskal-Wallis and ANOVA are "asymptotically equivalent" and so P-values are exactly the same for very large samples and they do not differ by much for small sample size.

Because of the equivalence, we can then expand nonparametric analysis based on ranks to any multi-factorial ANOVAs, regressions, MANOVA, ANCOVA, etc **NOTE:** Non-parametric tests are those that can handle non-normal data

There is a common misunderstanding in the statistical literature and among practitioners, including many biostatistics books, that non-parametric tests can also handle differences in variances among samples.

THIS IS NOT TRUE! They are also affected by variance differences among groups/treatments (i.e., homoscedasticity).

Test variance differences in ranks (almost never done in the literature)!

