Mathematical Statistics for Pedophiles

by Cyril E. Galaburda

October 2017

Abstract

This article is a textbook for those who study the "Meta-Analytic Examination of Assumed Properties of Child Sexual Abuse Using College Samples" (1998) by Bruce Rind, Philip Tromovitch and Robert Bauserman. The essential principles of probability theory, correlation analysis, and statistical tests theory are explained. Among which path analysis, variance analysis, regression analysis, contrast analysis, and sermi-partial correlational analysis are expounded.

More and more science influences our everyday lives. And purely academical calculations by Bruce Rind, Philip Tromovitch & Robert Bauserman (1998) [14] may influence our politics, morals, education, and privacy. As the scientists meta-analyzed 59 studies on child sex "abuse" experience in college students' childhood, and it is clear that keeping children in families is much more harmful and dangerous than adult-child sex itself!

What does it mean for us? It means that the right to privacy for children [19] tones with the right to privacy for pedophiles [20, 1]. It means that anti-pedophiliac genocide [5] must be stopped, and parents must be imprisoned for keeping children as their property, as slaves [17]. It means that child anti-sex abuse must become a crime, and if a child wants sex, the sex is by no means abuse.

Of course, Bruce Rind et al. are afraid to make such conclusions. Only pedophiles are in great need of the truth. That's why pedophiles must embalm the Meta-Analysis results. For this purpose they have to understand mathematical statistics and know how to reply to the moralists' insinuations.

My text is a fortification for pedophiles. Its basis is probability theory, its walls are statistical tests. They will withstand any moralists' lie about harmfulness of fornication, and one may fire back with correlation analysis.

I hope that truth will annihilate morals.

Population

Bruce Rind investigates adult-child sex "survivors". There have always been either 13-years-old prostitutes like Baron Corvo's boys, or 13-years-old clients of prostitutes like Federico Fellini, or sex fiends' preys like Mike Tyson, or 10-years-old wives like Mrinolini Debi, or fathers' victims like Joyce Meyer, or 12-years-old teacher-fuckers like

Vili Fualaau, or 14-years-old rock groupies like Lori Mattix, or poets'/philosophers'/sultans' minions like Abu Nuwas, or boys picked out for sex by savages like Pueblo Shamans, or "young members of the working class" shown in Paris baths to Jean Cocteau, or young porn-watchers like Luis Bunuel, or the others not known to us. The number of all the "survivors" of adult-child sex (so called "population") is infinite. What do they have in common with each other?

People believe, all the population shows one or another psychopathologic symptom. If we sample, say, $N(\text{sex})$ people from the population and find psychopathologic symptoms in $N(\text{symptom})$ of them these numbers are supposed to satisfy the equation:

$$\frac{N(\text{symptom})}{N(\text{sex})} = 1.$$

The left part of this equation may be called "conditional frequency of a symptom in adult-child sex survivors" or $f(\text{symptom}|\text{sex})$.

It is a fact that there are adult-child sex "survivors" for which

$$f(\text{symptom}|\text{sex}) \neq 1.$$

Moralists say that either such samples are too young to show any symptoms, or that psychometry isn't perfect, or that the samples had been enjoying the moral support in their families, or something else [18, 14]. So we cannot use frequencies and must take another quantity,

$$\lim_{N(\text{sex})\to\infty} f(\text{symptom}|\text{sex}) \equiv P(\text{symptom}|\text{sex}),$$

which is called "conditional probability of a symptom in adult-child sex survivors". Now, this quantity can say something about the population.

If we want to know, for instance, whether family environment reduces symptomatic rates (as moralists say) we should estimate probability of psychopathologic symptoms in different types of samples' families by probability multiplication:

$$P(\text{family \& symptom}|\text{sex}) = P(\text{family}|\text{sex})P(\text{symptom}|\text{sex}).$$

If we want to know whether PTSD is probable for our samples, we should sum up probabilities for all possible symptoms of PTSD,

$$\begin{aligned} P(\text{PTSD}|\text{sex}) = \\ P(\text{anxiety}|\text{sex}) + P(\text{flashbacks}|\text{sex}) + \\ P(\text{nightmares}|\text{sex}) + P(\text{insomnia}|\text{sex}) + \\ P(\text{irritability}|\text{sex}) + .., \end{aligned}$$

like anxiety, flashbacks, nightmares, insomnia, irritability and so on.

Also the more people we study the better we know whether it is probable to have a psychopathologic symptom without experience of adult-child sex:

$$P(\text{symptom} \setminus \text{sex}) = P(\text{symptom}) - P(\text{symptom \& sex}).$$

(Those who haven't had any adult-child sex experience in childhood are called "a control group".)

If, suppose, having a psychopathologic symptom does not depend on having experience of adult-child sex in childhood (the symptom is observed in adult-child sex "survivors" as often as in controls) we may say that having the symptom and having adult-child sex in childhood are "independent events" for which:

$$\begin{gathered} P(\text{symptom}) = P(\text{symptom}|\text{sex}) = \frac{P(\text{symptom \& sex})}{P(\text{sex})} \\ \Downarrow \\ P(\text{symptom \& sex}) = P(\text{symptom})P(\text{sex}). \end{gathered} \tag{1}$$

So if we know probabilities we may obtain whatever we want.

But how to obtain probabilities for our population? There is no way to obtain them. The population is infinite. But still probabilities can be and are used.

For that we must value our samples at different variable quantities. These are: "independent variables" of adult-child sex ξ, "dependent variables" for different psychopathologic symptoms η, and so called "third variables" ζ that characterize adult-child sex "survivors'" families at the time of the adult-child sex episode. For instance, we may take $\xi = 1$ for a sample if (s)he has experience of adult-child sex in childhood and $\xi = 0$ if not. This person may pass some psychological test and have η points of some psychopathologic symptom. If the person have grown without family problems let be $\zeta = \zeta_1$, if the person had been poor in childhood $\zeta = \zeta_2$, if the person had been neglected by parents $\zeta = \zeta_3$, and so on. These data

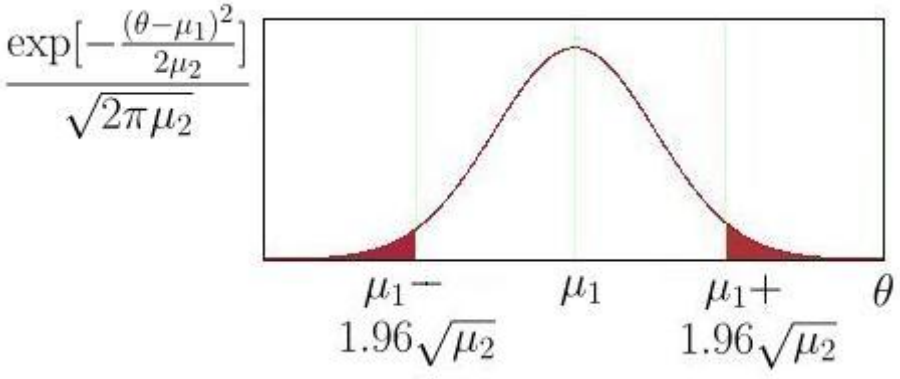

Figure 1: Normal Distribution. It is improbable for a normally-distributed quantity to be $\pm 1.96\sqrt{\mu_2}$ higher/lower than μ_1.

allow us to identify any sample and to obtain statistics for groups of samples.

For a variable like θ we use function of "probability distribution"

$$P(\theta < \theta_1) \equiv \Phi_\mu(\theta_1)$$

dependent on some parameter μ. To know this parameter for our population is to know everything about it.

What probability distribution functions are possible? According to the Limiting Theorems, if some variable $\theta_1 = \sum_n \theta_{1n}$ and $\forall\theta_{1n} \ll \theta_1$ this variable has "Normal Distribution":

$$\Phi_{\mu_1,\mu_2}(\theta_1) = \int_{-\infty}^{\theta_1} d\theta \frac{\exp[-\frac{(\theta-\mu_1)^2}{2\mu_2}]}{\sqrt{2\pi\mu_2}}$$

(Fig. 1). Another example. According to Karl Pearson (1900), a sum $\chi^2_L \equiv \sum_{l=1}^{L} \theta_l^2$ every θ_l of which has Normal Distribution $\Phi_{0,1}(\theta_l)$ is distributed as:

$$\Phi(\chi^2_L) = \int_0^{\chi^2_L} d\theta \frac{\theta^{\frac{L}{2}-1} \exp[-\frac{\theta}{2}]}{\sqrt{2}\Gamma(\frac{L}{2})}$$

where $\Gamma(\dots)$ is the Gamma Function.

There are also Exponential Distribution, Even Distribution, Beta Distribution, Student Distribution and many others, but still we don't know how the variables ξ, η and ζ are distributed.

What can we know about them? Any variable θ tends to have a value

$$\mu(\theta) \equiv \int_{-\infty}^{\infty} [\Phi_\mu(\theta) - \Phi_\mu(\theta - d\theta)]\theta$$

called "expected value", and their difference squared tends to have a value

$$\mu\left(\Delta^2\theta\right) \equiv \mu\left\{[\theta - \mu(\theta)]^2\right\}$$

called "variance".

For instance, a normally-distributed variable θ_1 is expected to be $\mu(\theta_1) = \mu_1$, and its $\mu\left(\Delta^2\theta\right) = \mu_2$. A Pearson-distributed variable χ^2_L is expected to be $\mu\left(\chi^2_L\right) = L$, and its $\mu\left(\Delta^2\chi^2_L\right) = 2L$.

It is easy to calculate that:

$$\begin{cases} \mu(\xi\eta) = \mu(\xi)\mu(\eta) \Leftrightarrow (1), \\ \mu\left(\Delta^2\theta\right) = \mu\left(\theta^2\right) - \mu^2(\theta), \\ \mu\left[\Delta^2(\theta_1+\theta_2)\right] = \mu\left(\Delta^2\theta_1\right) + \mu\left(\Delta^2\theta_2\right), \\ \mu\left[\Delta^2(C\theta)\right] = C^2\mu\left(\Delta^2\theta\right), \end{cases} \tag{2}$$

where C is some constant.

In the end we know that only expected and variance values of independent, dependent and third variables can say something about population.

Sampling

We want to study the population of adult-child sex "survivors". Neither Bruce Rind, nor Doctor Laura, nor FBI, nor anyone else can know their number. It is impossible to investigate all of them.

That's why we must sample some part of the population, investigate it and generalize from the part's qualities about the rest of the population.

The most popular adult-child sex "survivors" sampling methods are clinical sampling and legal sampling. However data obtained from these samples are not generalizable.

If we are talking about clinical sampling (to investigate only those adult-child sex "survivors" who resort to psychiatrical or psychologi-

cal care) it can be used to prove not only that sex causes psychopathologic symptoms, but also that masturbation causes psychopathologic symptoms. Yes, clinical samples were used by a Swiss physician Samuel Auguste Tissot (1728 – 1797) in showing that masturbation harmed. But according to Sir James Paget (1814 – 1899) Tissot's patients had been inclined to masturbation, not harmed by it. At the same time Tissot had been inclined to attribute psychopathologic symptoms in his patients to their experience of masturbation [10] like modern psychiatrists/psychologists are biased to attribute their patients'/clients' psychopathologic symptoms to their adult-child sex experiences [16]. Also many psychopathologic symptoms in masturbation survivors had been just arrogated to them because Tissot thought that masturbation had caused amnesia [10]. It is quite similar to modern search for "repressed memories" about adult-child sex in psychiatrists' patients and psychologists' clients. If we compare anti-masturbation ideology in XIX century and anti-sex ideology nowadays we may see that clinical sampling is not enough to show that masturbation/sex harms.

Are legal samples (those whose sex with adults have been disclosed by police) enough? According to anti-Rind criticism they aren't. Because police treats minor prostitutes like offenders, not victims; because adult-child sex "survivors" are afraid to make their experience known; because none would believe them and so on. That's why legal samples studies are said to be unreliable [18]. For only $\frac{1}{1000}$ part of American adult-child sex cases and $\frac{1}{3000}$ of European ones are

known to police [3]. Also if we investigate a legal sample we cannot say whether his/her psychopathologic symptoms have been caused by sex itself or by "secondary victimization" during parental interrogations, criminal & medical investigations, legal procedures and press intrusion. According to Candidate of Medical Science V. D. Badmaeva "for 10% of survivors (of adult-child sex) being under examination or at a trial is a deciding, trigger factor which influences more than sex offence itself" [2]. It is difficult to know how Ms. Badmaeva detected whether a survivor had been victimized, not secondary victimized, but according to another study "in 141 cases (of adult-child sex a psychiatrist) Körner found that in 10% the child had suffered physical or mental harm from the sexual activity — and in 29.2% harm had been inflicted by the the interrogation" [3]. So we can see that "protecting" children from sex causes psychopathologic symptoms at least as often as sex itself. Therefore legal sampling cannot prove that adult-child sex harms.

If we can use neither clinical, nor legal samples how can we sample adult-child sex "survivors" for investigation? A Dutch psychologist Fritz Bernard (1920 – 2006) which have funded scientific study of childlove used to send invitations for potential adult-child sex "survivors" throughout the country. This method is called "community sampling" and it shows that adult-child sex "survivors" "in community samples tend to be either normal or only slightly impaired on psychological measures" [14].

If we sample from the population so as to generalize from the

samples' qualities about all the adult-child sex "survivors" of some country it is called "national sampling". There had been studies on British, American and Spanish adult-child sex "survivors". All of them were meta-analysed by Bruce Rind et al. (1997) and shown that adult-child sex "survivors" have almost as many psychopathologic symptoms as those who haven't got such experience [12].

Community and national samples can really show a lot but for some college professor his/her students are much more handy for study. That's why student samples studies is "the largest group of studies on nonclinical populations,.. is useful for addressing questions regarding the general population because about 50% of U. S. adults have some college exposure" [14].

Anti-Rind critics objects saying that one cannot generalize from college students' mental resilience, from college students' luck not to have "serious" experience, from college students' lack of time to develop psychopathologic symptoms and another college students' qualities about the population of adult-child sex "survivors". But Bruce Rind et al. have shown (2001) that psychopathologic symptoms rates for college, pre-college, former college students and non-students are similar. So college students' qualities are quite generalizable [15].

Studying Students

Suppose we take N students and mark those of them who had had adult-child sex in childhood with the value $\xi = 1$. The rest of them are marked as $\xi = 0$. This way we get N values of the independent variable:

$$\xi = \xi_{i_1}, \qquad \xi = \xi_{i_2}, \qquad .., \qquad \xi = \xi_{i_n}, \qquad .., \qquad \xi = \xi_{i_N},$$

where $\forall i_n \in \{1, 2\}$.

Every n-th student passes some psychological test showing intensity of some psychopathologic symptom, and the student can get either η_1, or η_2, or .., or η_j, or .., or η_J points:

$$\eta = \eta_{j_1}, \qquad \eta = \eta_{j_2}, \qquad .., \qquad \eta = \eta_{j_n}, \qquad .., \qquad \eta = \eta_{j_N},$$

where $\forall j_n \in \{1, 2, .., j, .., J\}$.

Also we may test n-th student's family. If the student have grown without family problems let be $\zeta = \zeta_1$, if the student had been poor in childhood $\zeta = \zeta_2$, if the student had been neglected by parents $\zeta = \zeta_3$, .., if the student had been beaten by parents $\zeta = \zeta_M$. This way we get N values of third variable:

$$\zeta = \zeta_{m_1}, \qquad \zeta = \zeta_{m_2}, \qquad .., \qquad \zeta = \zeta_{m_n}, \qquad .., \qquad \zeta = \zeta_{m_N},$$

where $\forall m_n \in \{1, 2, .., m, .., M\}$.

Do $3N$ values of our variables prove that adult-child sex causes psychopathologic symptoms? It can be ascertained only through their weighted average values.

For any given variable $\theta \in \{\theta_1, \theta_2, .., \theta_l, .., \theta_L\}$ its "weighted average" $\langle\theta\rangle$ is defined as

$$\langle\theta\rangle \equiv \sum_{l=1}^{L} C_l \theta_l,$$

and its coefficients C_l are called "weights". If we want them to satisfy equations

$$\begin{cases} \lim\limits_{N\to\infty} \langle\theta\rangle = \mu(\theta), \\ \lim\limits_{N\to\infty} \left\langle \Delta^2\theta \right\rangle = \mu\left(\Delta^2\theta\right) \end{cases}$$

the weights must be $C_l = f(\theta = \theta_l)$ for $\langle\theta\rangle$, and $C_l = f(\theta = \theta_l)\frac{N}{N-1}$ for $\left\langle \Delta^2\theta \right\rangle$.

Suppose we know averages

$$\begin{cases} \langle\xi\rangle = \sum\limits_{i=1}^{2} \xi_i f(\xi = \xi_i) = \sum\limits_{n=1}^{N} \xi_{i_n} \dfrac{1}{N}, \\ \langle\eta\rangle = \sum\limits_{j=1}^{J} \eta_j f(\eta = \eta_j) = \sum\limits_{n=1}^{N} \eta_{j_n} \dfrac{1}{N}, \\ \langle\zeta\rangle = \sum\limits_{m=1}^{M} \zeta_m f(\zeta = \zeta_m) = \sum\limits_{n=1}^{N} \zeta_{m_n} \dfrac{1}{N}, \end{cases}$$

and samples variances

$$\begin{cases} \left\langle \Delta^2 \xi \right\rangle = \sum_{n=1}^{N} \left[\xi_{i_n} - \langle \xi \rangle \right]^2 \frac{1}{N-1}, \\ \left\langle \Delta^2 \eta \right\rangle = \sum_{n=1}^{N} \left[\eta_{j_n} - \langle \eta \rangle \right]^2 \frac{1}{N-1}, \\ \left\langle \Delta^2 \zeta \right\rangle = \sum_{n=1}^{N} \left[\zeta_{m_n} - \langle \zeta \rangle \right]^2 \frac{1}{N-1} \end{cases}$$

for our independent, dependent and third variables. Do these values prove that adult-child sex causes psychopathologic symptoms?

No, they do not. Whether η varies for the reason that $\Delta\xi \neq 0$, can be ascertained through their "Pearson correlation coefficient"

$$r(\xi, \eta) \equiv \frac{\langle \Delta\xi \Delta\eta \rangle}{\sqrt{\langle \Delta^2 \xi \rangle} \sqrt{\langle \Delta^2 \eta \rangle}}.$$

One may see that absolute value of Pearson correlation coefficient never exceeds one. When $r(\xi, \eta) = \pm 1.00$ our variables ξ and η are in functional dependence on each other which can (according to Taylor Series Theory) be presented as linear: $\eta = A_1 \xi + A_0$. [Because $\langle \Delta\xi A_1 \Delta\xi \rangle = A_1 \left\langle \Delta^2 \xi \right\rangle$ and $\sqrt{\langle \Delta^2 (A_1 \xi) \rangle} = |A_1| \sqrt{\langle \Delta^2 \xi \rangle}$.] The linear dependence means that increasing/decreasing ξ makes η increase/decrease too when $r(\xi, \eta) = \frac{A_1}{|A_1|} = 1.00$. On the other hand, increasing/decreasing ξ makes η decrease/increase if $r(\xi, \eta) = \frac{A_1}{|A_1|} = -1.00$.

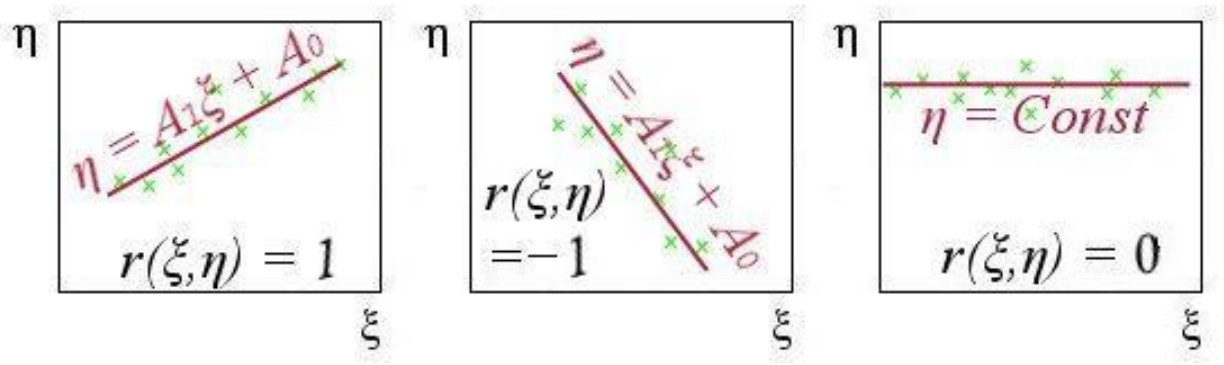

Figure 2: Meaning of Pearson ξ-η correlation coefficient.

In the case when $r(\xi, \eta) = 0.00 \Rightarrow \langle \Delta\xi \Delta\eta \rangle = 0$ we may say that while ξ varies ($\Delta\xi \neq 0$) η doesn't vary ($\Delta\eta = 0$), so ξ does not influence η, and the latter cannot be called "dependent" variable (Fig. 2).

Thus we can say that adult-child sex is concerned with psychopathologic symptoms if $r(\xi, \eta) = 1.00$, that psychopathologic symptoms are not caused by adult-child sex if $r(\xi, \eta) = 0.00$, that adult-child sex seems to prevent psychopathologic symptoms if $r(\xi, \eta) = -1.00$.

What Pearson correlation coefficients do student sampling studies show? For student samples

$$r(\xi, \eta) \in [-0.25, 0.40].$$

Adult-child sex and psychopathologic symptoms are almost non-associated. Adult-child sex is unlikely to produce psychopathologic

symptoms. For Bruce Rind and Philip Tromovitch "results of the meta-analyses indicate that, while about two out of 100 control individuals fall in the clinical range, about three out of 100 CSA individuals will" [13]. Harmless adult-child sex cannot be called "CSA".

This is not the whole story. We may say that our dependent variable η varies both along the line $\eta = A_1\xi + A_0$ and sideways the line:

$$\Delta\eta = \Delta(A_1\xi + A_0) + [\eta - (A_1\xi + A_0)].$$

According to (2)

$$\begin{cases} \left\langle \Delta^2\eta \right\rangle = A_1^2 \left\langle \Delta^2\xi \right\rangle + \left\langle [\eta - (A_1\xi + A_0)]^2 \right\rangle, \\ \langle \Delta\xi\Delta\eta \rangle = \langle \Delta\xi\Delta(A_1\xi + A_0) \rangle + \langle \Delta\xi[\eta - (A_1\xi + A_0)] \rangle = \\ \qquad A_1 \left\langle \Delta^2\xi \right\rangle, \end{cases}$$

because for any variable θ its variance is $\left\langle \Delta^2(\theta - \langle\theta\rangle) \right\rangle = \left\langle \Delta^2\Delta\theta \right\rangle = \left\langle \Delta^2\theta \right\rangle$ and variation $[\eta - (A_1\xi + A_0)]$ does not depend on variation $\Delta\xi$, so

$$\langle \Delta\xi[\eta - (A_1\xi + A_0)] \rangle \overset{(1)}{=} \langle \Delta\xi \rangle \langle \eta - (A_1\xi + A_0) \rangle = 0.$$

Therefore we can see that value

$$r^2(\xi, \eta) = \frac{\langle \Delta\xi\Delta\eta \rangle^2}{\left\langle \Delta^2\xi \right\rangle \left\langle \Delta^2\eta \right\rangle} = \frac{\left\langle \Delta^2\eta \right\rangle - \left\langle [\eta - (A_1\xi + A_0)]^2 \right\rangle}{\left\langle \Delta^2\eta \right\rangle}$$

shows what fraction of the variance $\left\langle \Delta^2\eta \right\rangle$ cannot be attributed to deviating η from the line $\eta = A_1\xi + A_0$. The value $r^2(\xi, \eta)$ shows

what fraction of the variance $\left\langle \Delta^2 \eta \right\rangle$ can be attributed to variation of our independent variable ξ. That's why $r^2(\xi, \eta)$ is coefficient of determining η by ξ.

How much psychopathologic symptoms are determined by adult-child sex experience? For students psychopathologic symptoms are nine times less determined by adult-child sex than by their families:

$$r^2(\zeta, \eta) \sim 9r^2(\xi, \eta),$$

so keeping children in families is nine times more harmful than sex with adults. Parents, not pedophiles, must be killed first!

Studying Studies

Bruce Rind, Philip Tromovitch, and Robert Bauserman have collected more than 54 papers on adult-child sex experience in college students. There they have found N_1 values of Pearson sex-symptoms correlation coefficients like

$$r = r_{k_1}, \qquad r = r_{k_2}, \qquad .., \qquad r = r_{k_n}, \qquad .., \qquad r = r_{k_{N_1}},$$

where $\forall k_n \in \{1, 2, .., k, .., K\}$ and $n \in \{1, 2, .., N_1\}$.

Among these N_1 values

$$\begin{gathered}
\text{a value } r = r_1 \text{ is found } N_1 f(r = r_1) \text{ times,} \\
\text{a value } r = r_2 \text{ is found } N_1 f(r = r_2) \text{ times,} \\
\vdots \\
\text{a value } r = r_k \text{ is found } N_1 f(r = r_k) \text{ times,} \\
\vdots \\
\text{a value } r = r_K \text{ is found } N_1 f(r = r_K) \text{ times.}
\end{gathered}$$

Our N_1 values of Pearson sex-symptoms correlation coefficient taken from 54 papers must be averaged. But how? According to Frans Gieles, "a correlation coefficient r ...is not an interval measure: i. e. the distance between $r = 0.1$ to $r = 0.2$ is not the same as the distance from $r = 0.8$ to $r = 0.9$" [6]. Before averaging we must convert our rs into some interval measures zs:

$$\begin{gathered}
z = \frac{1}{2} \ln \frac{1 + r_{k_1}}{1 - r_{k_1}}, \\
z = \frac{1}{2} \ln \frac{1 + r_{k_2}}{1 - r_{k_2}}, \\
\vdots \\
z = \frac{1}{2} \ln \frac{1 + r_{k_n}}{1 - r_{k_n}}, \\
\vdots \\
z = \frac{1}{2} \ln \frac{1 + r_{k_{N_1}}}{1 - r_{k_{N_1}}},
\end{gathered}$$

and average them like:

$$\begin{aligned}
\langle z \rangle &= \frac{1}{2} \ln \frac{1+r_1}{1-r_1} f(r=r_1), \\
\langle z \rangle &= \frac{1}{2} \ln \frac{1+r_2}{1-r_2} f(r=r_2), \\
&\vdots \\
\langle z \rangle &= \frac{1}{2} \ln \frac{1+r_k}{1-r_k} f(r=r_k), \\
&\vdots \\
\langle z \rangle &= \frac{1}{2} \ln \frac{1+r_K}{1-r_K} f(r=r_K),
\end{aligned}$$

or:

$$z_u = \sum_{n=1}^{N_1} \frac{1}{2N_1} \ln \frac{1+r_{k_n}}{1-r_{k_n}} = \frac{1}{2} \ln \frac{1+0.09}{1-0.09},$$

and get average Pearson coefficient for sex-symptoms correlation:

$$r_u = 0.09$$

which means that adult-child sex in childhood is barely associated with psychopathologic symptoms in adulthood.

Null Hypothesis

Mathematical statistics cannot be reduced to averaging variables like θ. One should test whether a samples average θ_u agrees with the

value $\mu(\theta)$ expected in the population. We have to know probability distribution function $\Phi_\mu(\theta)$ in the population of adult-child sex "survivors".

Suppose we've got some sample (of N students or N_1 student sampling studies) for which

$$\theta = \theta_{l_1}, \qquad \theta = \theta_{l_2}, \qquad .., \qquad \theta = \theta_{l_n}, \qquad .., \qquad \theta = \theta_{l_{N_{(1)}}}. \quad (3)$$

These values of θ are probable to be distributed either by $\Phi_\mu(\theta)$, or by $\Phi_{\mu'}(\theta)$, or by $\Phi_{\mu''}(\theta)$, or by another function. We cannot know exact function since our data are incomplete. If we expand or curtail the data, some distribution functions will seem more probable, and another ones will seem less probable for θ in the population.

We need only those distribution functions which probability exceeds some, so called p-value. Bruce Rind et al. (1998) often take $p \equiv 0.05$, and we say:

> Probability for θ to be distributed by one of the functions $\{\Phi_\mu(\theta), \Phi_{\mu'}(\theta), \Phi_{\mu''}(\theta), ..\}$ is "at the 0.05 level".

Such saying is called "Null Hypothesis". Null Hypothesis determines an ["$(1-p)\times 100\%$ confidence"] interval $\{\mu, \mu', \mu'', ..\}$, probability of membership in which for $\mu(\theta)$ exceeds p. If we estimate $\mu(\theta)$ at a value $\theta_u \notin \{\mu, \mu', \mu'', ..\}$ we should state another hypothesis that is "alternative" to Null Hypothesis. The samples data (3) may contradict our Null Hypothesis even when Null Hypothesis is correct. In this case we make an "Error of First Kind"

which probability is

$$P(\theta_u \notin \{\mu, \mu', \mu'', ..\}|\text{Null Hypothesis}) = p.$$

How to know whether our data (3) contradict Null Hypothesis? Sometimes it is enough to see whether positive/negative values of θ prevail over the values of the opposite sign in (3). Such prevalence really says something about distribution functions.

Another way is using a quantity (called "test statistic") which distribution function is known for us (for instance, it may be Normal Distribution). Depending on (3) the quantity takes probable or improbable values. If the value is improbable Null Hypothesis is dismissed.

Our Null Hypothesis will be:

> Psychopathologic symptoms in adulthood are caused by anything except adult-child sex.

Alternative Hypothesis: Homogeneity of Correlation Coefficients

If Null Hypothesis is correct different studies must result in discrepant values of Pearson sex-symptoms correlation coefficient:

$$\text{Null Hypothesis} = (r \in [-1.00, 1.00]).$$

But in reality "the resulting unbiased effect size estimate, based on 15,912 participants, was $r_u = 0.09$, with a 95% confidence interval from 0.08 to 0.11" [14]. If Null Hypothesis was correct such confidence interval would be less probable than Error of First Kind:

$$P\left\{r_u \in [0.08, 0.11] | r \in [-1.00, 1.00]\right\} = \frac{0.11 - 0.08}{1.00 - (-1.00)} = 0.015 < 0.050 = p.$$

That's why we must assume Alternative Hypothesis:

> Different studies result in homogeneous values of Pearson correlation coefficient.

But whether our results are really homogeneous? To clarify this we must know that if we've got N_1 values of Pearson sex-symptom correlation coefficients

$$r = r_{k_1}, \qquad r = r_{k_2}, \qquad .., \qquad r = r_{k_n}, \qquad .., \qquad r = r_{k_{N_1}},$$

among which

$$\begin{gathered}
\text{a value } r = r_1 \text{ is found } N_1 f(r = r_1) \text{ times,} \\
\text{a value } r = r_2 \text{ is found } N_1 f(r = r_2) \text{ times,} \\
\vdots \\
\text{a value } r = r_k \text{ is found } N_1 f(r = r_k) \text{ times,} \\
\vdots \\
\text{a value } r = r_K \text{ is found } N_1 f(r = r_K) \text{ times,}
\end{gathered}$$

a sum

$$\sum_{k=1}^{K} N_1 \frac{[f(r=r_k) - P\,(r=r_k|\text{Null Hypothesis})]^2}{P\,(r=r_k|\text{Null Hypothesis})} \qquad (4)$$

has variance

$$\mu\left\{\Delta^2 \sum_{k=1}^{K} N_1 \frac{[f(r=r_k) - P(r=r_k|\text{Null Hypothesis})]^2}{P(r=r_k|\text{Null Hypothesis})}\right\} = \mu\left(\Delta^2 \chi^2_{K-1}\right)$$

when $\sum_{(k\leq K)} 1/P(r=r_k|\text{Null Hypothesis}) \ll N_1$, and $K \ll N_1$ [11], and

$$P(r=r_k|\text{Null Hypothesis}) = P\left\{r_u \in [0.08, 0.11] | r \in [-1.00, 1.00]\right\} = 0.015.$$

So our test statistic here is χ^2_{K-1} that is supposed to equal (4) if Null Hypothesis is correct. Bruce Rind et al (1998) have calculated that

$$\sum_{k=1}^{54} N_1 \frac{[f(r=r_k) - P(r=r_k|\text{Null Hypothesis})]^2}{P(r=r_k|\text{Null Hypothesis})} = 78$$

which is quite improbable for $\chi^2_{54-1} = \chi^2_{53}$. This value is five times less probable than Error of First Kind:

$$P\{\chi^2_{53} \in [78, \infty)\} = 0.014 < 0.050 = p,$$

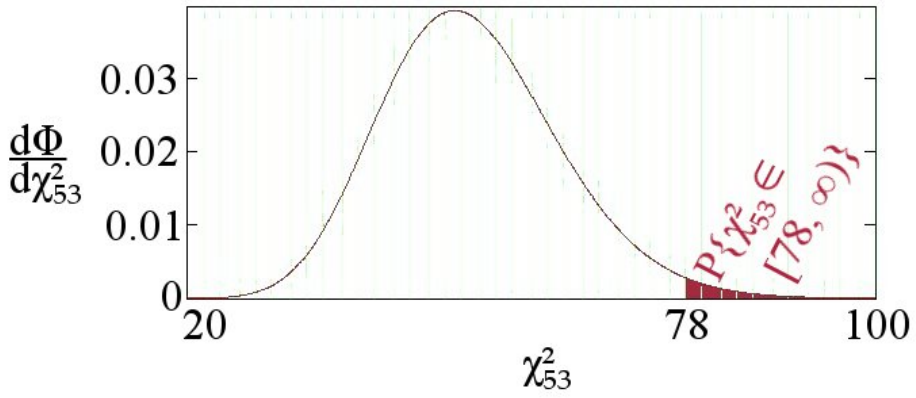

Figure 3: Distribution function for χ^2_{53}. The value $\chi^2_{53} = 78$ is improbable.

(Fig. 3) and we must keep our Null Hypothesis.

But Bruce Rind et al. (1998) know how to save rs homogeneity. They declared the values

$$r = 0.36, \qquad r = 0.40, \qquad r = -0.25, \tag{5}$$

for which

$$\langle z \rangle = 2.71, \qquad \langle z \rangle = 3.16, \qquad \langle z \rangle = -3.60$$

to be improbable (so called "outliers"). If we suppose that

$$\forall k_n \ : \langle z \rangle \gg \frac{1}{2} \ln \frac{1 + r_{k_n}}{1 - r_{k_n}} f(r = r_{k_n})$$

we may expect that $\langle z \rangle$ has Normal Distribution. And it is generally known that probability for any normally-distributed value $\langle z \rangle$ to

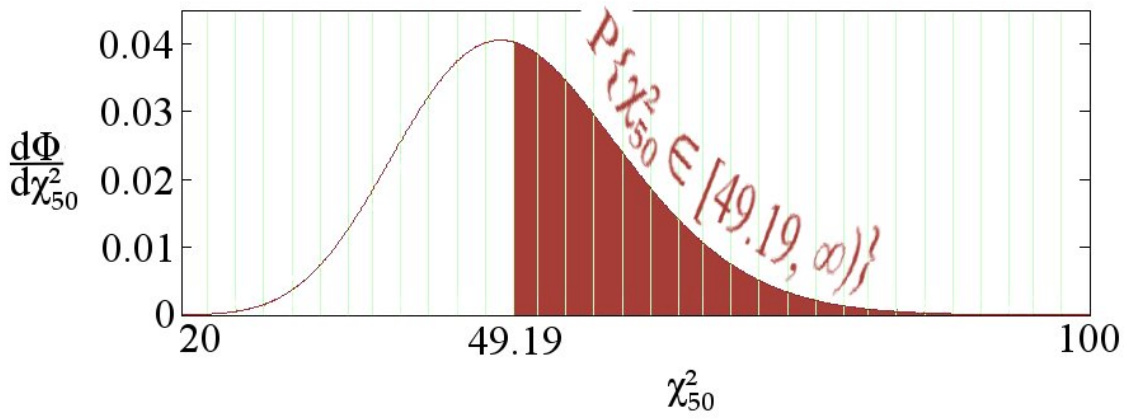

Figure 4: Distribution function for χ^2_{50}. The value $\chi^2_{50} = 49.19$ is probable.

be $1.96\sqrt{\langle\Delta^2\langle z\rangle\rangle}$ higher or $1.96\sqrt{\langle\Delta^2\langle z\rangle\rangle}$ lower than it's expected value z_u is less than 0.05:

$$P\left(|\Delta\langle z\rangle| > 1.96\sqrt{\langle\Delta^2\langle z\rangle\rangle}\right) \leq 0.05.$$

That's why Bruce Rind et al. (1998) ignore the values (5) and have

$$\sum_{k=1}^{51} N_1 \frac{[f(r = r_k) - P(r = r_k|\text{Null Hypothesis})]^2}{P(r = r_k|\text{Null Hypothesis})} = 49.19$$

which is quite probable for $\chi^2_{53-3} = \chi^2_{50}$:

$$P\{\chi^2_{50} \in [49.19, \infty)\} = 0.506 \gg 0.050 = p$$

(Fig. 4).

That's why Bruce Rind et al. (1998) prefer Alternative Hypothesis to Null Hypothesis.

Alternative Hypothesis: Sex is Worse Than Domestic Violence

The fact that adult-child sex correlates with psychopathologic symptoms does not mean that symptoms are caused by sex itself. "While correlations may sometimes provide valuable clues in uncovering causal relationships among variables, a non-zero estimated correlation between two variables is not, on its own, evidence that changing the value of one variable would result in changes in the values of other variables. For example, the practice of carrying matches (or a lighter) is correlated with incidence of lung cancer, but carrying matches does not cause cancer" [4]. In this example fluctuations of "independent" (carrying matches) and dependent (cancer) variables are caused by fluctuations of third variable (smoking).

In the same way Bruce Rind et al. (1998) suppose that psychopathologic symptoms (dependent variable) and sex life ("independent variable") are both caused by non-sexual abuse in childhood (third variable). Children are sexual beings only in the case when they've got bad parents.

For me it's rubbish. Mohandas Gandhi married when he was 13-

years-old not because he'd got problems with parents. But abusive parents may drive a child to psychopathologic symptoms when they punish him/her for sex.

That's why we may word our Null Hypothesis as:

> Family, not sex, causes psychopathologic symptoms,

and Alternative Hypothesis as:

> It is better to be poor/ignored/railed/threaten/beaten than satisfied.

These hypotheses weren't tested by Bruce Rind et al. (1998) personally, just taken from another papers on "statistical control". That was accomplished with path analysis, variance analysis and regression analysis.

Let's start from path analysis [21]. It supposes that fluctuations of our independent, dependent and third variables are associated with points of a directed graph, any influence on some fluctuation is associated with a rib of the graph, and any path in the graph is associated with a linear combination of fluctuations which are supposed to influence each other.

In this model our Null Hypothesis will look like:

$$\Delta\eta = A_{23}\Delta\zeta + A_{21}\Delta\xi,$$

and Alternative Hypothesis will look like:

$$\Delta\eta \neq A_{23}\Delta\zeta + A_{21}\Delta\xi.$$

This linear combination may be divided by square root of dependent variable variance:

$$\frac{\Delta\eta}{\sqrt{\langle\Delta^2\eta\rangle}} = \left(\frac{A_{23}\sqrt{\langle\Delta^2\zeta\rangle}}{\sqrt{\langle\Delta^2\eta\rangle}}\right)\frac{\Delta\zeta}{\sqrt{\langle\Delta^2\zeta\rangle}} + \left(\frac{A_{21}\sqrt{v\langle\Delta^2\xi\rangle}}{\sqrt{\langle\Delta^2\eta\rangle}}\right)\frac{\Delta\xi}{\langle\Delta^2\xi\rangle}. \tag{6}$$

Numerators between parantheses are square roots of dependent variable variance when all the other variables but one do not fluctuate:

$$\begin{cases} A_{23}\sqrt{\langle\Delta^2\zeta\rangle} = \sqrt{\langle\Delta^2\eta\rangle}\Big|_{\xi=\mathrm{Const}}, \\ A_{21}\sqrt{\langle\Delta^2\xi\rangle} = \sqrt{\langle\Delta^2\eta\rangle}\Big|_{\zeta=\mathrm{Const}}. \end{cases}$$

If we multiply (6) by independent variable fluctuation and average it, we will see from (2) that

$$r(\xi,\eta) = \left(\frac{\sqrt{\langle\Delta^2\eta\rangle}\Big|_{\xi=\mathrm{Const}}}{\sqrt{\langle\Delta^2\eta\rangle}}\right) r(\zeta,\eta) + \left(\frac{\sqrt{\langle\Delta^2\eta\rangle}\Big|_{\zeta=\mathrm{Const}}}{\sqrt{\langle\Delta^2\eta\rangle}}\right)$$

when Null Hypothesis is correct. If we know Pearson coefficients of sex-symptom correlation, family-sex correlation and family-symptom correlation (they are 0.09, 0.13 and 0.29 respectively which means that keeping children in families is much more harmful than sex with them); if we know variances of symptom variable under different values of the other variables, path analysis will show whether Null

Hypothesis is really correct. This method shown (1988) that incest-symptom correlations for girls are non-significant without analysing family background of incest [8].

That's all I must say about path analysis, and now we'll study variance analysis [7]. Suppose we've got N_0 students that have grown without family problems ($\zeta = \zeta_1$), N_0 students that have been poor in childhood ($\zeta = \zeta_2$), N_0 students that have been neglected by parents ($\zeta = \zeta_3$), .., N_0 students that have been beaten by parents ($\zeta = \zeta_M$). All the students are sorted into M groups, and intensity of some psychopathologic symptom in them are evaluated as

$$\begin{array}{lllll}
\eta = \eta_{j_{11}}, & \eta = \eta_{j_{21}}, & .., & \eta = \eta_{j_{m1}} & .., \quad \eta = \eta_{j_{M1}}, \\
\eta = \eta_{j_{12}}, & \eta = \eta_{j_{22}}, & .., & \eta = \eta_{j_{m2}} & .., \quad \eta = \eta_{j_{M2}}, \\
\vdots & \vdots & & \vdots & \vdots \\
\eta = \eta_{j_{1n}}, & \eta = \eta_{j_{2n}}, & .., & \eta = \eta_{j_{mn}} & .., \quad \eta = \eta_{j_{Mn}}, \\
\vdots & \vdots & & \vdots & \vdots \\
\eta = \eta_{j_{1N_0}}, & \eta = \eta_{j_{2N_0}}, & .., & \eta = \eta_{j_{mN_0}} & .., \quad \eta = \eta_{j_{MN_0}}.
\end{array}$$

We may average these values like

$$\langle\eta\rangle = \langle\eta\rangle_1, \quad \langle\eta\rangle = \langle\eta\rangle_2, \quad .., \quad \langle\eta\rangle = \langle\eta\rangle_m, \quad .., \quad \langle\eta\rangle = \langle\eta\rangle_M$$

or like

$$\mu(\eta) = \sum_{m=1}^{M} \sum_{n=1}^{N_0} \eta_{j_{mn}} \frac{1}{MN_0}.$$

Any fluctuation of the symptom variable like

$$\Delta\eta_{j_{mn}} = \left[\sum_{m=1}^{M}\eta_{j_{mn}} - \langle\eta\rangle_m\right] + \left[\sum_{m=1}^{M}\langle\eta\rangle_m - \mu(\eta)\right]$$

contains of varying on account of family and varying on account of another causes. (2) and

$$\sum_{n=1}^{N_0}\left(\eta_{j_{mn}} - \langle\eta\rangle_m\right) = \left(\sum_{n=1}^{N_0}\eta_{j_{mn}}\right) - N_0\langle\eta\rangle_m = \left(\sum_{n=1}^{N_0}\eta_{j_{mn}}\right) - N_0\sum_{n=1}^{N_0}\eta_{j_{mn}}\frac{1}{N_0} = 0$$

mean that total variance

$$\left\langle\Delta^2\eta\right\rangle = \left\langle\Delta^2\eta|_{\zeta=\mathrm{Const}}\right\rangle + \left\langle\Delta^2\langle\eta\rangle\right\rangle$$

is sum of "within-group variance" and "external variance", that shows whether family environment influences intensity of symptoms in adult-child sex "survivors".

One may see that

$$\mu\left[\Delta^2\eta|_{\zeta=\mathrm{Const}}\right] = \mu\left[\eta^2 - 2\eta\langle\eta\rangle + \langle\eta\rangle^2\right] = \mu\left[\langle\eta\rangle^2 - 2\langle\eta\rangle\mu(\eta) + \mu^2(\eta)\right] = \mu\left(\Delta^2\langle\eta\rangle\right),$$

so while

$$\frac{\Delta\eta|_{\zeta=\text{Const}}}{\sqrt{\mu\left(\Delta^2\eta|_{\zeta=\text{Const}}\right)}} = \frac{\Delta\eta|_{\zeta\ =\text{Const}}}{\sqrt{\mu\left(\Delta^2\langle\eta\rangle\right)}} \qquad \text{and} \qquad \frac{\Delta\langle\eta\rangle}{\sqrt{\mu\left(\Delta^2\langle\eta\rangle\right)}}$$

have Normal Distribution $\Phi_{0,1}$

$$(MN_0 - N_0)\frac{\left\langle\Delta^2\eta|_{\zeta=\text{Const}}\right\rangle}{\mu\left(\Delta^2\langle\eta\rangle\right)}$$

has the same distribution function as $\chi^2_{MN_0-N_0}$,

$$(M-1)\frac{\left\langle\Delta^2\langle\eta\rangle\right\rangle}{\mu\left(\Delta^2\langle\eta\rangle\right)}$$

has the same distribution function as χ^2_{M-1}, and variances quotient

$$\frac{\left\langle\Delta^2\langle\eta\rangle\right\rangle}{\left\langle\Delta^2\eta|_{\zeta=\text{Const}}\right\rangle}$$

has the same distribution function as

$$F(M-1, MN_0 - N_0) \equiv \frac{\chi^2_{M-1}/(M-1)}{\chi^2_{MN_0-N_0}/(MN_0 - N_0)}.$$

The F is distributed in a predictable way, and may be used as a test statistic which corroborates our Null Hypothesis.

That's all I must say about variance analysis, and now we'll study regression analysis. It supposes the Null Hypothesis to mean

$$\langle\eta\rangle = B_1\zeta + B_0,$$

where coefficients

$$\begin{cases} B_1 = r(\zeta, \eta)\dfrac{\sqrt{\langle \Delta^2 \eta \rangle}}{\sqrt{\langle \Delta^2 \zeta \rangle}}, \\ B_0 = \langle \eta \rangle - \langle \zeta \rangle B_1 \end{cases}$$

are calculated from empirical data. If empirical data contradict the idea of linear subjection between $\langle \eta \rangle$ and ζ than the Alternative Hypothesis:

$$\langle \eta \rangle = A_1 \xi + A_0,$$

may be correct. But it didn't happen.

It had been shown through path analysis, variance analysis and regression analysis that a quarter to all of sex-symptom correlations are caused by non-comfortable family environments of adult-child sex "survivors". That's why living in a family harms, and sex does not harm. Children must be protected from parents, not from pedophiles.

Correlation Coefficients as Dependent Variables

Investigating whether sex-symptom correlation coefficients depend on third variables like θ we may consider them as independent variables, and the correlation coefficients as dependent variables.

Suppose possible values of θ are

$$\theta = \theta_1, \qquad \theta = \theta_2, \qquad .., \qquad \theta = \theta_l, \qquad .., \qquad \theta = \theta_L,$$

and we've got L groups of Pearson sex-symptom correlation coefficient values with N_0 values per group and $\theta = \theta_l$ in any l-th group:

$$\begin{array}{lllll}
r = r_{k_{11}}, & r = r_{k_{21}}, & .., & r = r_{k_{l1}} & .., \quad r = r_{k_{L1}}, \\
r = r_{k_{12}}, & r = r_{k_{22}}, & .., & r = r_{k_{l2}} & .., \quad r = r_{k_{L2}}, \\
\vdots & \vdots & & \vdots & \vdots \\
r = r_{k_{1n}}, & r = r_{k_{2n}}, & .., & r = r_{k_{ln}} & .., \quad r = r_{k_{Ln}}, \\
\vdots & \vdots & & \vdots & \vdots \\
r = r_{k_{1N_0}}, & r = r_{k_{2N_0}}, & .., & r = r_{k_{lN_0}} & .., \quad r = r_{k_{LN_0}}.
\end{array}$$

These values may be converted into zs:

$$\begin{array}{lllll}
z = z_{11}, & z = z_{21}, & .., & z = z_{l1} & .., \quad z = z_{L1}, \\
z = z_{12}, & z = z_{22}, & .., & z = z_{l2} & .., \quad z = z_{L2}, \\
\vdots & \vdots & & \vdots & \vdots \\
z = z_{1n}, & z = z_{2n}, & .., & z = z_{ln} & .., \quad z = z_{Ln}, \\
\vdots & \vdots & & \vdots & \vdots \\
z = z_{1N_0}, & z = z_{2N_0}, & .., & z = z_{lN_0} & .., \quad z = z_{LN_0},
\end{array}$$

and averaged like

$$\langle z \rangle = \langle z \rangle_1, \quad \langle z \rangle = \langle z \rangle_2, \quad .., \quad \langle z \rangle = \langle z \rangle_l, \quad .., \quad \langle z \rangle = \langle z \rangle_L.$$

How to know whether these values were influenced by third variable? There are contrast analysis and sermi-partial correlational analysis to clarify it.

Contrast analysis supposes that if θ influenced sex-symptom correlation coefficients there would be

$$\begin{cases} C_1\langle z\rangle_1 + C_2\langle z\rangle_2 + ... + C_l\langle z\rangle_l + ... + C_L\langle z\rangle_L = 0, \\ C_1 + C_2 + ... + C_l + ... + C_L = 0. \end{cases}$$

In the case of $L = 2$, for instance, it means that either for $\theta = \theta_1$, or for $\theta = \theta_2$ correlation coefficients would be the same

$$\begin{gathered} C_1\langle z\rangle_1 + C_2\langle z\rangle_2 = \langle z\rangle_1 - \langle z\rangle_2 = 0 \\ \Downarrow \\ \langle z\rangle_1 = \langle z\rangle_2, \end{gathered}$$

and θ does not influence correlation anyhow.

Whether it is really so should be checked through F-test. Because

$$\frac{\Delta\langle z\rangle}{\sqrt{\mu\left(\Delta^2\langle z\rangle\right)}}$$

has Normal Distribution $\Phi_{0,1}$,

$$\frac{\Delta\sum_{l=1}^{L} C_l\langle z\rangle_l}{\sqrt{\mu\left(\Delta^2\sum_{l'=1}^{L} C_{l'}\langle z\rangle_{l'}\right)}} \overset{(2)}{=} \frac{\Delta\sum_{l=1}^{L} C_l\langle z\rangle_l}{\sqrt{\mu\left(\Delta^2\langle z\rangle\right)}}\sqrt{\frac{N_0}{\sum_{l'=1}^{L} C_{l'}^2}}$$

has Normal Distribution $\Phi_{0,1}$,

$$\frac{\left\langle \Delta^2\langle z\rangle \right\rangle}{\mu\left(\Delta^2\langle z\rangle\right)}(L-1)$$

has the same distribution function as χ^2_{L-1},

$$\frac{\left\langle \Delta^2 \sum_{l=1}^{L} C_l\langle z\rangle_l \right\rangle}{\mu\left(\Delta^2\langle z\rangle\right)}(LN_0-N_0)\times\frac{N_0}{\sum_{l'=1}^{L} C_{l'}^2}$$

has the same distribution function as $\chi^2_{LN_0-N_0}$,

$$\frac{\left\langle \Delta^2\langle z\rangle \right\rangle}{\left\langle \Delta^2 \sum_{l=1}^{L} C_l\langle z\rangle_l \right\rangle}\times\frac{\sum_{l'=1}^{L} C_{l'}^2}{N_0}$$

has the same distribution function as $F(L-1, LN_0-N_0)$ [7].

That's all I must say about contrast analysis, and now we'll study semi-partial correlational analysis. Semi-partial correlation is "the correlation between the dependent variable and the residual of the prediction of one independent variable by the other ones" [9], while residual is difference between the predicted independent variable and its prediction through another variables. "To predict" is to offer some functional dependence between the variables which (according to Taylor Series Theory) may be presented as linear.

In our case let dependent variable be $\langle z\rangle$, predicted independent variable be θ, predicting independent variable be ζ, prediction be

$$\langle\theta\rangle\equiv D_1\zeta + D_0.$$

Whether such prediction is correct must be checked through F-test again. The value of F conforms to semi-partial correlation coefficient

$$r\left[\langle z\rangle, \langle\theta\rangle - (D_1\zeta + D_0)\right] = r\left[\langle\theta\rangle - (D_1\zeta + D_0), \langle z\rangle\right].$$

How? Coefficient of determination "is expressed as the ratio of the explained variance (variance of the model's predictions...) to the total variance (sample variance of the dependent variable...)" [4], and any variance obeys rules (2), so

$$r^2[\langle\theta\rangle - (D_1\zeta + D_0), \langle z\rangle] = r^2[\langle\theta\rangle, \langle z\rangle] + r^2[\zeta, \langle z\rangle] = r^2[\zeta, \langle z\rangle],$$

since correlation coefficient may depend on variable θ, not on its average $\langle\theta\rangle$ which is constant. That's why Bruce Rind et al. (1998) tried "to obtain correlations between each moderator and the effect sizes" [14], not between the residual and the effect sizes.

One may see that

$$r[\zeta, \langle z\rangle]$$

has Normal Distribution $\Phi_{0,1}$,

$$\frac{\theta - (D_1\zeta + D_0)}{\sqrt{\mu\left(\Delta^2\theta\right)}}$$

has Normal Distribution $\Phi_{0,1}$,

$$r^2\left[\zeta, \langle z\rangle\right]$$

has the same distribution function as χ^2_1,

$$\frac{\left\langle [\theta - (D_1\zeta + D_0)]^2 \right\rangle}{\mu\left(\Delta^2\theta\right)}(N_0 - 2) = \left\{1 - r^2[\theta, D_1\zeta + D_0]\right\}(N_0 - 2)$$

has the same distribution function as $\chi^2_{N_0-2}$,

$$\frac{r^2[\zeta, \langle z\rangle]}{1 - r^2[\theta, D_1\zeta + D_0]}$$

has the same distribution function as $F(1, N_0 - 2)$ [9].

This way Bruce Rind et al. (1998) have found that such (third or independent, no matter) value as level of contact during adult-child sex does not influence sex-symptom correlations at all. The value which really influences is level of consent. That's why children can consent to sex (either with themselves, or with another children, or with adults), and if adult-child sex is wanted by the child it is harmless.

Conclusions

Here is what Bruce Rind et al. (1998) Meta-Analysis about. One, who understands this article, may estimate the Meta-Analysis at its true worth.

Bruce Rind may be blamed in pedophilia but passionless numbers cannot be pedophiliac. The American Psychological Association

may be forced to repudiate Bruce Rind but χ^2 cannot be forced to increase. It is possible to prohibit Scott Lilienfeld from writing about Bruce Rind but it is not possible to prohibit the Central Limit Theorem.

Not only common sense but also sexology (see Kinsey Reports), ethology (of Bonobos), ethnography (of Trobriands and another Australoids, of Papuans, of Sumatrans, of Muria, of Lepcha, of Northern Africans, of Hopi & Siriono Indians), history (of Ancient Greece, of Medieval Arabs & Persians, of Western Pre-Industrial societies), biographies (Guy Hocquenghem's, Kirk Douglas'es, Hans van Maanen's, and anothers), and finally mathematical statistics show that sex even with adults is harmless for children. It is the truth that shall make you free.

References

[1] American Convention on Human Rights, §11.

[2] Badmaeva, V. D., "Consequences of Sexual Abuse in Children and Adolescences," in Russian, *Zh. Nevrol., Psikhiatr. Im. S.S. Korsakova,* 2009, 109, 12, ISSN 1997-7298, P. 35.

[3] Brongersma, E., "Loving Boys," vol. 2, 1990, ISBN 1-55741-003-08.

[4] "Coefficient of determination," from *Wikipedia,* 2017, URL `https://en.wikipedia.org/w/index.php?title=Coefficient_of_determination&oldid=755104809`.

[5] "Genocide definitions," from *Wikipedia,* 2015, URL `https://en.wikipedia.org/w/index.php?title=Genocide_definitions&oldid=685915956`.

[6] Gieles, F., An Explanation of the Statistics used in the Meta-Analysis, IPCE NewsLetter, E7, 1999.

[7] Glass, V. G., Stanley, J. C., "Statistical Methods in Education and Psychology," in Russian, Moscow, 1976.

[8] Harter, S., Alexander, P., & Neimeyer, R., "Long-term Effects of Incestuous Child Abuse in College Women," *Journal of Consulting and Clinical Psychology,* 1988, 56, 5 – 8.

[9] Hervé Abdi, "Part (Semi Partial) and Partial Regression Coefficients," in: N. Salkind (ed.), *Encyclopedia of Measurement and Statistics,* 2007.

[10] Norlik, M., "Tabu-Zone: Wissenschaftliche Erkenntnisse und etische Grundsätze zum Umgang mit kindlicher Sexualität und Pädophilie", 2013.

[11] Pearson K., Experimental Discussion of the (χ^2, p) Test for Goodness-Of-Fit, *"Biometrika,"* 1932, vol. 24, P. 351 – 381.

[12] Rind, B., & Tromovitch, P., A Meta-Analytic Review of Findings from National Samples on Psychological Correlates of Child Sexual Abuse, *Journal of Sex Research,* 1997:34, Pp. 237 – 255.

[13] Rind, B., & Tromovitch, P., "National Samples, Sexual Abuse in Childhood, and Adjustment in Adulthood," *Archives of Sexual Behavior,* 2006.

[14] Rind, B., Tromovitch, Ph., Bauserman, R., A Meta-Analytic Examination of Assumed Properties of Child Sexual Abuse Using College Samples, *Psychological Bulletin,* 1998, vol. 124, No. 1, Pp. 22 – 53.

[15] Rind, B., Tromovitch, Ph., Bauserman, R., "The Validity and Appropriateness..," *Psychological Bulletin,* 2001, vol. 127, No. 6.

[16] Rivas, T., Positive Memories, 1st print, 2013, ISBN / EAN: 978-90-815403-1-5, Case BW-06.

[17] Slavery Convention, §1.

[18] Thice, P. P., et al., 2008, URL `https://vk.com/doc10086728_442359757?hash=c1b93db763ece73cec&dl=63643d0fadf36b79ee`.

[19] The UN Convention on the Rights of the Child, §§16, 40¶2.

[20] The UN Universal Declaration of Human Rights, §12.

[21] Wright, S., "The Method of Path Coefficients," *The Annals of Mathematical Statistics,* vol. 5, No. 3, 1934, Pp. 161 – 215.