THE UNIVERSITY of ADELAIDE

Robinson Research Institute

PO Box 3151, Melbourne St, North Adelaide

21 April 2017

Mr R Brent Wisner
Baum, Hedlund, Aristei & Goldman
12100 Wilshire Blvd., Suite 950
Los Angeles, CA 90025

Dear Mr Wisner

Thank you for asking me to provide a critical appraisal of the four citalopram/ escitalopram randomized controlled trials (RCT) in children and adolescents.

I am a child psychiatrist and full professor in the Disciplines of Psychiatry and Paediatrics at the University of Adelaide. I head Adelaide University's Critical and Ethical Mental Health research group (CEMH), which conducts research, teaching and advocacy in order to promote safer, more effective and more ethical research and practice in mental health; and the Paediatric Mental Health Training Unit (PMHTU), which provides training and support to medical students, GPs, allied health professionals, teachers and counsellors in non-pathologising approaches to primary care mental health.

I have occupied senior clinical roles in child psychiatry for 30 years, and have been teaching, researching and publishing in the field of critical appraisal and research methodology for two decades. I have acted as an expert witness in many courts, including the Australian Supreme Court.

My attached CV (Appendix 3) contains details of my many publications, several of them highly cited, in the area of research methodology and reporting, particularly in relation to antidepressants. Those publications that are most relevant to this task are bolded in Appendix 3.

In order to prepare this report, I requested copies of original protocols and any protocol amendments on the clinical study reports (both drafts and final versions), published papers and any available drafts of those papers for all four RCTs. In addition, you provided me with a range of other documents as outlined in Appendix 2. I have considered all of those documents that you made available to me judged to be relevant to appraising the studies, but I have not, for example, read the depositions in detail. All of my analyses take account of all the information examined. I am happy to be directed towards any document that may appear to contradict my expressed opinion. Except where otherwise indicated, sources below are the relevant clinical study reports (CSR) and published papers for each study.

Citalopram Study 94404

Study 94404, a 12-week RCT comparing citalopram (10-40 mg per day) and placebo in the treatment of major depression in adolescents, was conducted by Lundbeck in 31 centres in 7 European countries between 19 November 1996 and 23 April 2001. The stated objective was "to study the efficacy and tolerability of citalopram compared to placebo in adolescent patients suffering from major depression". 120 patients were randomised to placebo (112 treated) and 124 to citalopram (121 treated). 38 placebo (34%), and 42 citalopram treated patients withdrew, while 17 (15%) of placebo and 22 (18%) of citalopram patients had serious adverse events (SAE). The level of withdrawal and adverse events (AE) was much higher for this study than the other three, reflecting two important methodological advantages of 94404: longer duration (12 vs 8 weeks) and the inclusion of patients with a history of suicidal activities or thought.

There was no numerical (let alone statistically significant) efficacy advantage to citalopram over the placebo response. With regard to harms, we cannot be confident about the level of AEs without access to individual patient level (IPL) data. However the CSR reports a clinically significant difference in suicide attempts, with 5 (4.5%) in the placebo group and 14 (11.6%) in the citalopram group.[1]

[1] CSR, panel 25, p72

Looking at those patients for whom there were patient narratives in the CSR (p285) confirms a more worrying profile for citalopram than placebo:

Table 1: Adverse Events from patient narratives*

	Citalopram	Placebo
Any psychiatric AE	**27**	**12**
Deliberate self-harm or suicidal thinking/behaviour that was classified as serious, and/or led to withdrawal	**22**	**6**
Significant suicidal behaviour	**12** (007, 009, 121, 426, 573, 761, 776, 874, 874, 884, 884, 884)	**2** (412, 871)

* including post study events but not screening failures. One patient allocated to placebo took an overdose on citalopram prescribed after the study, and is here counted in the citalopram group. In addition one citalopram patient (577) had QT prolongation, now known to be a significant danger of the drug.

In my opinion, 94404 was a more appropriately designed study than MD-18 as it most closely approximated real-world use (longer duration of treatment and inclusion of suicidal patients), and was larger and restricted to adolescents who are more likely to have been responsive to the drug. Forest has claimed that the US study (MD-18) was a more accurate study than the European 94404, in part because analysis could not be adjusted for differences in whether the patients had a more complicated depressive disorder.[2] First, I note the failure of MD-18 to effectively control for prior exposure to antidepressants, thus effectively failing to adjust for some patients having "a more complicated depressive disorder." As noted below, it is not clear whether or not this asymmetry between the two groups would significantly bias results, but it does undermine arguments that MD-18 was "cleaner."

Second, as Lundbeck argues in an email apparently from David John Simpson, dated 5 Nov 2004, the European study was more rigorous than US studies (in this case, MD-15, which had similar methodology to MD-18 and MD-32, see Appendix 1) because "whereas the European study included patients as one would be likely to encounter in daily life, and therefore was actually a better study in terms of treatment 'effectiveness' the USA trial applied more stringent

[2] Press Release, Forest Laboratories, Inc., *Forest Discusses Disclosure of Citalopram Clinical Trials Data in Children and Adolescents* (June 24, 2004)

inclusion criteria and in that sense resembles classic clinical trials with 'ideal' patients no one ever sees on one's doorstep."

Citalopram Study CIT-MD-18

MD-18 was conducted between 31 Jan 2000 and 10 Apr 2001, and was designed as a 9-week, 20-site, comparison of the safety and efficacy of citalopram versus placebo in children (age 7–11) and adolescents (age 12–17) with major depressive disorder. The study design included a 1-week, single-blind placebo lead-in followed by an 8-week, double-blind treatment phase. The study protocol specified that the primary efficacy measure was the change from baseline to week 8 on the Children's Depression Rating Scale-Revised (CDRS-R) total score.

MD-18 was not a statistically or clinically positive study, though it was misleadingly reported to be. Forest violated ethical standards required of a drug manufacturer in the conduct, publication, and promotion of MD-18 in a number of ways:

1. *Failure to report and appropriately deal with unblinding*

Nine patients were treated with either pink citalopram or white placebo, breaking the blind for the investigators, who were informed of the unblinding. It also broke the blind for those five patients receiving citalopram, who would have received plain white, then "trade-dress" pink, then plain white tablets. Note that whether or not the patients themselves were aware, unblinding is still deemed to have occurred if the investigators might have become aware of whether or not the patient has received medication or placebo. Dr. Paul Tiseo,[3] acknowledged: "dispensing these tablets would automatically unblind the study."[4]

[3] Dr. Tiseo was Forest's primary monitor of MD-18, and Dr. Flicker was his supervisor.

[4] In preparing this report, I have seen new relevant documentation that raises the possibility that the breaking of the blind for investigators could possibly have extended beyond the 9 patients who were dispensed medication prior to recalling the trade-dress drug. Document MDL-FORP0206957-8, a Forest Laboratories memo from Irene Green, March 8, 2000, and Document MDL-FORP0206959-60, a Forest Laboratories Deviation Report show that *all* 640 bottles of citalopram tablets to be used for all citalopram patients in the first weeks of the study were "trade-dress" (ie pink and recognizable as active drug). These packs were opened to confirm that they contained trade-dress medication, and replaced with non-trade-

The mean decrease in CDRS for the unblinded citalopram group was 30.5, higher than the 21.3 for blinded patients.

Table 2: Unblinded patients in MD-18

Patient no.	treatment group	age group	CDRS baseline	CDRS week 8	difference in CDRS
105	citalopram	child	84	17	67
114	citalopram	child	70	18	52
505	citalopram	adol	82	-	-
506	citalopram	adol	79	84	+5
513	citalopram	adol	63	55	8
citalopram mean decrease in CDRS		30.5 (vs 21.3 for blinded patients)			
113	placebo	child	92	56	36
507	placebo	adol	55	49	6
514	placebo	adol	92	77*	15
509	placebo	adol	67	49	18
placebo mean decrease in CDRS		18.75 (vs 16.4 for blinded patients)			

*LOCF from week 6

Appendix Table 6 of the MD-18 Study Report shows a primary outcome calculation excluding these patients.[5] This per protocol exclusion resulted in a statistically "negative" primary efficacy outcome. In reporting the study however, eight of the excluded patients were included in the analysis, turning the (albeit marginally) statistically insignificant outcome ($p < 0.052$) into a statistically significant outcome ($p < 0.038$). The unblinding error was not reported in the published article.

There are at least three scientific and ethical problems here:

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dress. MDL-FOREM0010335 indicates that while only the nine patients had been randomised at the point where the drug packaging error was noted, most sites had already been sent randomised drugs.

No statement is made as to whether or how the blind was protected in the process of replacing the trade-dress drug. The documents state that the relevant medication "was quarantined" but not that the inevitable unblinding of those who made the substitution was not shared with anyone. I have not found enough detail in any of the documents to be confident about the details of that randomisation process. It is possible that medication was not yet allocated to a particular patient. If this was so, then there would be less concern about those staff who replaced the trade-dress medication with the plain white tablets knowing which was which. But if not, information about which packs did and did not contain citalopram could have reached others in a way that broke the blind. Absent any reassurance that this unblinding could not have occurred, the study should have been terminated or all patients should have been re-randomised.

[5] In my previous analysis of study MD-18, I noted that Appendix Table 6 predated CSR Table 3.1. At the time, I understood this to mean that the analysis including unblinded patients had preceded that excluding them. Further information since provided to me makes it clear that an analysis including the unblinded patients predated Appendix Table 6. Therefore, the possibility raised by me that the initial analysis has been deliberately suppressed is no longer supported by the information available to me. This observation does not however alter the main conclusions about unblinding as outlined here.

i. *Failure to accurately report in the CSR, and to openly disclose to the FDA.*

Forest seemingly deliberately downplayed the unblinding in reporting it to the FDA. Amy Rubin, who worked in Forest Regulatory Affairs, changed a draft of the FDA letter from stating that the dispensing error could have "unblinded the study" to stating that the dispensing error had the "potential to cause patient bias."[6] Although Dr. Charles Flicker objected that "the integrity of the blind was unmistakenly violated" the submitted letter was unaltered in this respect.

When it came to writing the CSR, an earlier draft (MDL-FORP0018664-730) shows handwritten edits, apparently introduced by Dr Flicker. These edits have the effect of playing down the importance of unblinding, for example:

- by replacing "unblinded study drug treatment" with "medication with potentially unblinding information" in "Nine patients (Patients 105, 113, 114, 505, 506, 507, 509, 513, and 514) were mistakenly dispensed 1 week of medication with potentially unblinding information (tablets had an incorrect color coating)." (CSR, p63.)
- by replacing "who accidentally received 1 week of unblinded study drug treatment" with "for whom the study blind was potentially compromised" in "Appendix Table 6 presents the results from the LOCF analysis for the change from baseline to Week 8 excluding data from the 9 patients for whom the study blind was potentially compromised (see Section 5.3.4). The results from the Week 8 LOCF analysis comparing the mean change from baseline in CDRS-R in the citalopram and placebo groups was not substantially affected by the exclusion of those patients; the LSM difference decreased from 4.6 to 4.3 and the p-value increased from 0.038 to 0.052." (CSR, p70.)
- by adding a paragraph on "Validity" that includes another downplay of the importance of unblinding: "A medication packaging error partially compromised the study blind for 9 of the 174 patients. Post-hoc analysis excluding these patients supported the results from the intent-to-treat analysis. It is concluded that the study results are valid and interpretable." (CSR, p83.)

Subsequent to Dr Flicker's edits, further downplaying of the importance of

[6] MDL-FOREM0030382

unblinding included the addition of the phrase "although otherwise blinded" to the sentence: "Because of a drug packaging error, the citalopram or placebo tablets initially dispensed to 9 patients at 3 study centers were distinguishable in color, although otherwise blinded (see Section 7.0). When this error was identified at the beginning of the study period, all study medication shipments were replaced in full with tablets of identical color to remove any potential for unblinding." (CSR, p44.)

Similarly, the notes from a conference call with Pharmanet in October 4, 2001 demonstrate an imperative to spin the data, with the term *secondary post-hoc analysis of the ITT subpopulation* coined to describe the analysis without unblinded patients (which should have been the primary analysis).

ii. *Including the unblinded patients in the reported primary analysis, thereby contravening the study's own protocol, ordinary scientific practice, and undertakings made to the FDA*.

The protocol for study MD-18 stipulated: "Any patient for whom the blind has been broken will immediately be discontinued from the study and no further efficacy evaluations will be performed" and Forest's advice to the FDA appropriately signalled that "the primary efficacy analysis will exclude the potentially unblinded patients." As has been acknowledged by Forest staff (eg, depositions of Charles Flicker and William Heydorn), the failure to exclude unblinded patients was a clear breach of protocol. None of the various rationalisations for including the unblinded patients carry any weight.

iii. *Failure to report the unblinding in the published article*.

In whatever manner the data were to be analysed and reported, Forest were obliged to declare the unblinding in their journal paper and other publications and presentations.

2. *Reporting spurious effect size*

A further exaggeration of the effect of citalopram was to report an effect size on the primary outcome measure of 2.9, a claim at odds with the primary data even with the unblinded patients included. The origin of the effect size calculation

remains unclear, with no reference to its calculation in the study protocol or the published paper. Wagner et al. publicly acknowledged an error and stated that "with Cohen's method, the effect size was 0.32"[7] but did not explain the initial misrepresentation.

3. *Failure to publish negative secondary outcomes, and undeclared inclusion of post hoc outcome measures*

While CGI-S and CGI-I were correctly reported in the published article, Wagner et al. failed to publish two of the protocol-specified secondary outcomes[8], both of which were unfavourable to citalopram (see p1081). This was deliberate. On October 15, 2001, Ms. Prescott wrote: "I"ve heard through the grapevine that not all the data look as great as the primary outcome data. For these reasons (speed and greater control) I think it makes sense to prepare a draft in-house that can then be provided to Karen Wagner (or whomever) for review and comments."[9] Subsequently, Forest's Dr. Heydorn wrote on April 17, 2002: "The publications committee discussed target journals, and recommended that the paper be submitted to the American Journal of Psychiatry as a Brief Report. The rationale for this was the following: . . . As a Brief Report, we feel we can avoid mentioning the lack of statistically significant positive effects at week 8 or study termination for secondary endpoints"[10]. Instead the writers presented post hoc statistically positive results that were not part of the original study protocol or its amendment (visit-by-visit comparison of CDRS-R scores, and "Response", defined as a score of ≤28 on the CDRS-R) as though they were protocol-specified outcomes. In particular, "Response" was reported in the results section of the Wagner et al. article after the primary but before the secondary outcomes, likely predisposing a reader to regard it as more important than the selected secondary measures reported, or even to mistake it for a primary measure (a strategy used with success in GSK's Study 329).

[7] Wagner KD, Robb AS, Findling RL, Jin J, Dr. Wagner and colleagues reply. Am J Psych. 2005;162(4):819.
[8] Kiddie Schedule for Affective Disorders and Schizophrenia-Present (depression module) and the Children's Global Assessment Scale (CGAS).
[9] E-mail re: Pediatric data dated 10/15/01.
[10] E-mail by Forest Staff re Peds Manuscript dated 4/17/02.

Notes from a conference call with Pharmanet October 4, 2001 confirm that the intent was to spin the data, with a statement that the 'secondary responder analysis (the percent of patient showing >= 50% decrease in the CDRS-R and K-SADS-P)" should, "if supportive" be included "in discussion of primary efficacy parameter".[11]

When it came to writing the CSR, Dr Flicker's edits have the effect of downplaying the fact that the secondary outcome measures did not show advantage to citalopram over placebo (see, for example, MDL-FOR0018714). He also eliminated from what became Panel 11 (CSR, p70), "Change from Baseline to Week 8 in CDRS-R (Mean ± SEM)" the Median and range, both of which showed citalopram in a less favourable light.

4. *Inadequate reporting of ineffective randomization*

It is a requirement of reporting RCTs that the process and outcome of randomisation be documented.[12] Whenever there is a notable discrepancy between the drug and placebo groups, any possible effects on outcomes should be canvassed.[13] 19 citalopram patients and 16 placebo patients had previous exposure to antidepressant medication documented.[14] Several patients had had more than one antidepressant medication or more than one prescription of the same medication. I divided the patients into three groups: those that had only positive response to one or more antidepressant prescriptions; those who had both positive and negative responses to different prescriptions; and those who had only poor responses to one or more medications. Notably, six citalopram patients versus one placebo patient had only a positive response to one or more antidepressant in the past. Otherwise figures were similar: four citalopram and five placebo patients had had a mixed response and nine citalopram and ten placebo patients had had only a negative response. It is not clear whether this

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[11] Strategies for minimising the impact of the failure to reach statistical significance on the secondary efficacy variables were also discussed during that conference call.

[12] CONSORT (CONsolidated Standards of Reporting Trials) 2010 guideline. http://www.consort-statement.org/consort-2010.

[13] Moher D, Hopewell S, Schulz KF, Montori V, Gøtzsche PC, Devereaux PJ, Elbourne D, Egger M, Altman DG; Consolidated Standards of Reporting Trials Group. CONSORT 2010 Explanation and Elaboration: Updated guidelines for reporting parallel group randomised trials. J Clin Epidemiol. 2010 Aug;63(8):e1-37.

[14] MD-18 CSR, p 1186, listing 6, 9/4/2001

had a significant biasing effect on the outcomes but were it to have an effect, it would be in favour of citalopram.

Furthermore, in his redraft of the CSR, Dr Flicker eliminated a paragraph accompanying what became panel 9 of the CSR (p66) that acknowledged that randomisation had had the result that 'statistical significantly (p=0.028) more Caucasian adolescents were enrolled in the citalopram group". While applying p values to randomisation results is inappropriate, researchers should clearly identify any possibly meaningful baseline discrepancies, and include discussion of what the implications might be for the results.[15] This finding, although of uncertain significance for the study, should nevertheless have been reported, and its possible implications discussed.

5. *Mischaracterisation of Adverse Events*

Although Wagner et al. correctly reported that "the rate of discontinuation due to AEs among citalopram-treated patients was comparable to that of placebo," the authors failed to mention that the five citalopram-treated patients discontinuing treatment did so due to one case of hypomania, two of agitation, and one of akathisia. None of these potentially dangerous states of over-arousal occurred with placebo. Furthermore, anxiety occurred in one citalopram patient (and none on placebo) of sufficient severity to temporarily stop the drug and irritability occurred in three citalopram (compared to one placebo). Taken together, these AEs raise concerns about dangers from the activating effects of citalopram that should have been reported and discussed. Instead Wagner et al. reported "adverse events associated with behavioral activation (such as insomnia or agitation) were not prevalent in this trial" and claimed that "there were no reports of mania" without acknowledging the case of hypomania.

Furthermore, there were many more gastrointestinal AEs for citalopram than placebo patients. However, Wagner et al. grouped the AE data in a way that in effect masked this possibly clinically significantly gastrointestinal intolerance.

[15]Moher D, Hopewell S, Schulz KF, Montori V, Gøtzsche PC, Devereaux PJ, Elbourne D, Egger M, Altman DG; Consolidated Standards of Reporting Trials Group. CONSORT 2010 Explanation and Elaboration: Updated guidelines for reporting parallel group randomised trials. J Clin Epidemiol. 2010 Aug;63(8):e1-37.

Finally, the published article also failed to report that one patient on citalopram developed abnormal liver function tests.

A further cause for concern about AE ascertainment in this study is the finding that "No sexual dysfunction was reported" (CSR, p77). Given that around a third of adults experience sexual difficulties with citalopram[16], this is either a striking finding that deserves considerable discussion, or, more likely, a failure in AE ascertainment. If sexual AEs were not elicited, this raises concern that that collection of data about other AEs may have been deficient. In any event, failing to collect information about sexual dysfunction results in an underestimate of harms.

6. *Priority of marketing over science/patient welfare*

While it is legitimate for sponsors to use positive RCTs to promote their products, the priority is to dispassionately inform the scientific community of the outcomes.[17] There is no doubt that the broadcast of the results of MD-18 was more of a marketing exercise than a scientific sharing of information. In a press release dated June 24 2004, Forest claimed that citalopram "is not approved for use in children, adolescents and has not been promoted by Forest for use in these populations"[18] but Exhibit 58 leaves no doubt that citalopram was being promoted off-label by drug reps between 1999 and 2002. Christina Goetjen, from Forest product management wrote in September 2001 of MD-18: "If we get this data presented in late January, we can use it while we"re still promoting Celexa. If we wait for it to be presented at publication, it will be long past its prime." [19] John McPhee, another Forest marketing executive, wrote: "I believe that ACNP does not allow the referencing of presentations made at their meeting. This could eliminate the ability to reference the presentation in slides used in a CME program. . . . If it is true, we need to find another venue to present the data so

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[16] Clayton AH, Croft HA, Handiwala L. Antidepressants and sexual dysfunction: mechanisms and clinical implications. Postgrad Med. 2014 Mar;126(2):91-9. doi: 10.3810/pgm.2014.03.2744.

[17] Doshi P, Dickersin K, Healy D, Vedula S, Jefferson T. Restoring invisible and abandoned trials: a call for people to publish the findings. BMJ 2013;346:f2865

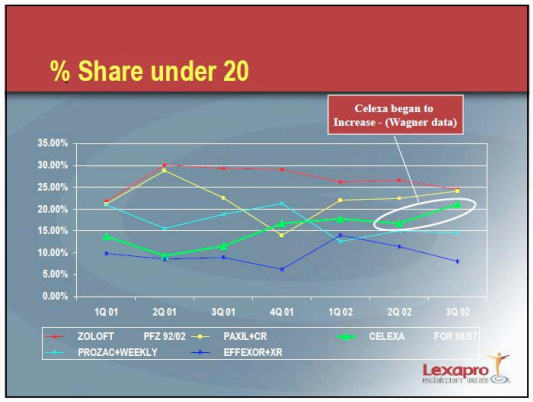
[18] Press Release, Forest Laboratories, Inc., *Forest Discusses Disclosure of Citalopram Clinical Trials Data in Children and Adolescents* (June 24, 2004). The claim in this press release that there was "no increased risk of suicidality" is clearly at odds with the findings of study 94404.

[19] Mitchner Email Re. ACCAP Meeting Friday, November 2, 2001

that it is referencable . . . make sure that a poster presentation is fair game for inclusion in the program / presentation . . . the poster must be written with a CME program in mind."

There is no evidence that I have seen that Dr Wagner played a significant part in the design or conduct of study MD-18 beyond being the manager of one of the sites where patients were collected. On the other hand, there are several references that show that she took a leadership role in marketing Celexa. Goetjen wrote on Oct 31 2001: "We spoke with Karen Wagner today about the current state of affairs regarding the pediatric data. We discussed Forest's decision to go with a publication other than JAMA as it fits with our corporate objectives. She agreed with the logic, yet reminded us that if we want to appeal to the PCP and Pediatric audiences, we need to publish in a place that provided the appropriate readership . . . She also said that the lack of data regarding the use of Celexa for pediatrics is limiting it to "last choice" among physicians - she just wanted to make sure we understood the marketing advantages of the data. I assured her we got it. She is excited about our Pediatric Regional CME series and will be a fundamental part of speaker selection. . . . She is extremely savvy about PR and is working well with GCI for surrounding PR opportunities." McPhee responded the same day: "Thanks -- my feeling is that the fact that we are last for ped use is the very reason we can"t wait to disseminate data until JAMA publishes." On Nov 1, Goetjen wrote: "We must have the manuscript to Dr. Wagner as soon as possible to push the publication . . . she understands the urgency to get this data in front of our audience as soon as possible if we"re going to maximize the impact."

Forest appeared happy with Dr Wagner's performance:[20]



7. *Ghostwriting*

There are clear guidelines for what constitutes authorship of a study and both ghostwriting (unacknowledged authorship) and "guest" authorship (named authors who make insufficient contribution) are proscribed.[21] Of the several ethical concerns about ghostwriting, the most relevant to patient welfare is its use to give priority to a marketing message over science and the welfare of patients. The misrepresentation of MD-18 was facilitated by the ghostwriting of the published article and other materials. Control over content and management of the article resided with Forest. Dr. Heydorn (Forest Senior Study Director) wrote to Mr. Lawrence on October 15, 2001: "Given what I have seen of the data, I believe that we should maintain control, which means either writing in house or having an outside group (like Weber Shandwick [BSMG] or a CRO) draft the manuscript."[22] The MD-18 manuscript was prepared by Natasha Mitchner at Weber Shandwick Communications, under instruction from Jeffrey Lawrence (Product Manager Forest Marketing) before the academic "authors" were chosen.[23]

Escitalopram Study ESC-MD-15

Forest Research Institute's MD-15 "A Double-Blind, Placebo-Controlled Evaluation of the Safety and Efficacy of Escitalopram in Pediatric Depression"

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[20] Forest Pharmaceuticals, Inc., F*Y'04 Lexapro Strategic Operations* (Dec. 3, 2002)

[21] International Committee of Medical Journal Editors (ICMJE) http://www.icmje.org/recommendations/browse/roles-and-responsibilities/defining-the-role-of-authors-and-contributors.html

[22] E-mail re: Pediatric data dated 10/16/01.

[23] E-mail by Forest Staff re Peds Manuscript dated 4/17/02; E-mail re: Pediatric data dated 10/15/01.

was conducted in 25 US centers from December 09, 2002 to February 06, 2004, with the stated primary aim "To evaluate the safety and efficacy of escitalopram in pediatric outpatients (6-17 years) diagnosed with major depressive disorder." It was a flexible dose study of 264 patients (268 patients were randomized), 6-17 years of age, who received at least one dose of double-blind study medication.

With regard to efficacy, there is no doubt that "This study failed to demonstrate the effectiveness of escitalopram 10-20 mg/day relative to placebo with respect to the primary endpoint, the change from Baseline to Week 8 in CDRS-R score using the LOCF approach."[24] The CSR attempts to mitigate this unwelcome outcome, by stating the following:

> "However, a trend toward significance was noted in two secondary efficacy parameters, CGI-S and CGAS, using the LOCF approach, supported by statistical significance using the OC approach."

This statement is factually correct. However the sentence that follows is phrased in a way that seems deliberately misleading:

> "An examination of results by age group demonstrated a significantly greater improvement with escitalopram relative to placebo among adolescents (12-17 years) in all primary and secondary efficacy measures except the LOCF analysis of CDRS-R."[25]

The primary outcome measure *is* the LOCF analysis of CDRS-R; thus the phrasing distracts the reader from the finding that the post hoc primary outcome measure was negative. All the other "positive" findings need to be judged in light of that.

Similarly in the published version of the study, the results are presented in a more favourable light than is justified. Although in the body of the paper it is acknowledged that "the trial was not powered to specifically detect efficacy in each age subgroup", the abstract (likely to be the most read part of the paper) states: "Escitalopram did not significantly improve CDRS-R scores compared to placebo at endpoint ... In a post hoc analysis of adolescent (ages 12-17 years) completers, escitalopram significantly improved CDRS-R scores compared with

[24] CSR synopsis
[25] 6.3.3 Additional Efficacy Analyses. "Additional efficacy parameters were: CDRS-R response rate (CDRS-R ≤ 28) at Week 8, and CGI-I response rate (CGI-I ≤ 2) at Week 8" but neither of these specified in protocol.

placebo." Again the negative primary outcome measure even in the post hoc group is suppressed.

The appropriate way to report this study, including the findings on the post hoc group[26] would be:

> "This study failed to demonstrate the effectiveness of escitalopram 10-20 mg/day relative to placebo with respect to the primary endpoint, the change from Baseline to Week 8 in CDRS-R score using the LOCF approach. *Similarly a post hoc analysis of results by age group failed to reach statistical significance for the adolescent sub-population on the primary outcome. However, some statistically significant outcomes were noted in secondary and post hoc efficacy parameters."*

Of concern is that published reviews uncritically cite such misrepresented conclusions, thus compounding the misleading effect on readers. For example, Carandang et al[27] incorrectly note that "post hoc analysis of study completers 12–17 years of age found a significant difference in favour of escitalopram on CDRS-R scores (p=0.047)". Carandang et al also quote the Wagner et al claim for an effect size of 2.9 in their report of study MD-18.

Furthermore, there are issues in the randomisation process that may have skewed results in favour of escitalopram. First, there were more patients who had failed previous trials of antidepressants in the placebo (27) than the citalopram group (17); and many more in the citalopram group (22) than the placebo group (3) had previously responded to antidepressants.[28] Second, "ongoing psychiatric comorbidity was reported in 17 patients (12.8%) in the placebo group and 8 patients (6.1%) in the escitalopram group" (CSR, p57); psychiatric comorbidity is associated with worse outcomes for adolescent

[26] Panel 13 (Change from Baseline to Week 8 in Efficacy Parameters in Adolescents, 12-17 Years (Mean ± SEM) — ITT Population, CSR, p61

[27] Carandang C, Jabbal R, Macbride A, Elbe D. A review of escitalopram and citalopram in child and adolescent depression. J Can Acad Child Adolesc Psychiatry. 2011 Nov;20(4):315-24.

[28] "Thirty patients (23%) in the placebo group and 39 patients (30%) in the escitalopram group had previously received antidepressant treatment; 27 of the 30 placebo-treated patients and 17 of the 39 escitalopram-treated patients had a history of treatment non-response. Relative to each treatment population, the percentage of previous non-responders was greater in the placebo group (20%) than in the escitalopram group (13%).", CSR, p57

depression.

With regard to harms, overall psychiatric AEs were surprisingly low (14.3% for placebo and 13.7% for drug), raising concerns about the collection of AE data. As noted above, we cannot be confident about the level of AEs without access to IPL (individual patient level) data, and therefore cannot be reassured by the apparent lack of excess AEs in the escitalopram group. An indication of tolerability comes from the overall withdrawal rate; for placebo this was 18 (13.5%) and for escitalopram 29 (22.1%).[29] In view of minimal information about the reasons for discontinuation[30], this can be cautiously taken as being suggestive of tolerability problems with escitalopram.

Escitalopram Study ESC-MD-32

MD-32 was a "Flexible-Dose Study of Escitalopram in Pediatric Patients With Major Depressive Disorder" conducted from April 1, 2005 - May 31, 2007. 316 patients were randomized, with 157 in the escitalopram safety population and 154 for placebo. According to FDA criteria, MD-32 was a positive study, because, unlike any of the other studies of (es)citalopram, it achieved statistical significance on its primary outcome measure. However there are several reasons to question the clinical significance of that outcome.

1. *Sample size*

In assessing the efficacy of a drug, it is often assumed that the larger the RCT, the more meaningful a statistically positive outcome will be. In fact, a smaller RCT that is well designed and conducted and proves to be statistically positive is a stronger indication of efficacy than the same P value in a larger trial. Conversely, a larger RCT that shows a statistically negative outcome is a strong indication of a lack of efficacy, since greater sample size increases the likelihood that a clinically insignificant benefit will show statistical significance.

[29] Total Withdrawn for Any Reason Placebo 18 (13.5%) Escitalopram 29 (22.1%) (Panel 8. Number (%) of Patients Who Prematurely Discontinued From the Study and Reason for Discontinuation — Safety Population)
[30] PATIENT DATA LISTINGS, LISTING 1 Patient Disposition

So a statistically negative outcome in a large trial (as was the case with 94404 and MD-15) more strongly supports a lack of efficacy than a statistically positive result in a trial of similar size supports efficacy (MD-32). There was no well-conducted small trial with a statistically positive outcome for (es)citalopram.

2. *Single Blind Run-in*

This trial, as with MD-18, used a one-week single blind run-in. Those excluded through a single blind run-in would otherwise have been randomly distributed to each group and more likely to meet criteria for efficacy at the end of the study. Therefore their exclusion is likely to strengthen the apparent superiority of drug over placebo, potentially misleading prescribers. Nowhere in the CSR is it disclosed how many patients were excluded after the single-blind; such patients were not identifiable within a total of 201 (34.4% of those screened) who "Did Not Meet Criteria"[31] so that it is unclear what impact removing these patients had on the overall outcome.[32]

3. *Statistical but not clinical significance*

There was a statistically significant difference between the baseline scores for the two treatment groups at baseline indicating greater depression severity in the escitalopram group: CDRS-R total score: Placebo 56.0 ± 8.3 vs Escitalopram 57.6 ± 8.3, p = .034). As noted above, it is inappropriate to use statistical significance in this setting, and the real question is whether the difference makes a clinically significant impact on the outcome. There are at least two problems with the published paper statement that: "nevertheless, these differences were not clinically significant."[33] First, the higher the initial score, the greater the

[31] Table 14.1.2. Reason for Screen Failure. Screened Population, CSR p111

[32] There is a further ethical concern about single-blind lead in methodology in terms of informed consent. Nowhere in the protocol for this study in is it specified what the patients were to be told about the one-week single blind run-in ("Patients who meet the eligibility criteria at Visit 2 will be dispensed one bottle (Bottle A) containing 10 placebo tablets. Patients will be instructed to take one tablet daily in the evening, starting on the day the medication is dispensed", protocol, p27, "10.2.1 Single-Blind Treatment"). Possibly patients were told they may or may not be receiving medication, so that from their perspective they were on the same medication in this first week as subsequently. But if so, they were mislead, since the person providing that information knew that they were receiving placebo, and such deception is argued to be unethical. (Evans M Justified deception? The single blind placebo in drug research Journal of Medical Ethics 2000;26:188-193)

[33] Emslie GJ1, Ventura D, Korotzer A, Tourkodimitris S. Escitalopram in the treatment of adolescent depression: a randomized placebo-controlled multisite trial. J Am Acad Child Adolesc Psychiatry. 2009 Jul;48(7):721-9. doi: 10.1097/CHI.0b013e3181a2b304.

reduction in score due to regression to the mean. Second, as a peer reviewer notes, where a difference of 1.6 on a scale with a range from 17 to 113 is rightly regarded as not being clinically significant, this must call into question the clinical meaningfulness of a difference in the primary efficacy outcome that is variously reported as 4 in the CSR (change at Week 8 (LOCF): Placebo –18.4 ± 1.1; Escitalopram –22.4 ± 1.1; p= .022), and 3.3 in the published study (Placebo –18.8 ± 1.27; Escitalopram –22.1 ± 1.22; p= 0.22).

The National Institute of Clinical Excellence (NICE)[34] regards a change of three points on the Hamilton Depression Rating Scale (HAM-D) as clinically significant.[35] The range on that scale is 0 to 68, so that by crude comparison with the 96 point range of the CDRS-R, 3-4 points difference falls short of clinical significance.

Similarly the effect of escitalopram was weak, as noted by a reviewer (low effect size of 0.27), raising further doubts about the clinical significance of the findings. In response to this review, the published paper offers a rationale that this effect size is no different from other antidepressants in youth, with the clear implication that the reader should be reassured, rather than the more neutral conclusion that it supports the clinical insignificance of any benefit from most or all antidepressants in youth.

4. *Underreporting of Adverse Events*

Another significant and dangerous distortion in the published reporting of MD-32 is the misrepresentation of adverse outcomes.

i. It is correctly reported in the published paper that two placebo and four escitalopram patients had SAEs. However one of these two placebo patients (0323202) only developed suicidal behaviour after ceasing placebo and starting on open label escitalopram.

[34] National Institute for Health and Clinical Excellence, Depression: Management of Depression in Primary and Secondary Care. Clinical Practice Guideline Number 23, National Institute for Clinical Excellence, London, 2004.

[35] There are coherent arguments to suggest that three points on the HAM-D actually falls well short of clinical significance, see Moncrieff J, Kirsch I. Empirically derived criteria cast doubt on the clinical significance of antidepressant-placebo differences. Contemp Clin Trials. 2015 Jul;43:60-2

ii. The published paper claimed that the "rate of discontinuation because of AEs did not differ for placebo (1 patient; 0.6%) versus escitalopram (4 patients; 2.6%; p = .21)". First, the use of statistical significance to judge the clinical significance of AEs is inappropriate. Secondly, according to the CSR[36], the event attributed to placebo in the published study in fact occurred in a patient who was not randomised (0413201) and should not have been included in the placebo category; only three such withdrawals are listed for escitalopram.

iii. The published paper fails to make clear that for the three escitalopram patients withdrawn due to protocol violation, (0213205, 0213206, and 0333203), "noncompliance with study drug and/or visits was reported by the Investigator at the time of discontinuation"[37] indicating possible tolerability issues.

Thus a table similar to the following should have been published to indicate possible harms from the medication.

Table 3: Adverse events in MD-32

	Placebo	Escitalopram
Serious AE	1	4*
Withdrawal because of AE	0	3
Withdrawal because of non-compliance	0	3

* 5, if emergence of suicidal actions on open label escitalopram is included

Reservations about the clinical significance of the efficacy results, the potential dangers of the drug and knowledge of three other negative studies should have led Forest to make much more cautious claims for the use of escitalopram, and should not have been used to license the medication for children.

Conclusion

(Es)citalopram is not clinically effective for child or adolescent depression, and its harms outweigh its benefits. No clinician should have been encouraged to prescribe (es)citalopram. As noted by Carandang et al[38], the FDA decision to

[36] CSR, p277, LIST OF PATIENT NARRATIVES

[37] CSR, p56

[38] Carandang C, Jabbal R, Macbride A, Elbe D. A review of escitalopram and citalopram in child and adolescent depression. J Can Acad Child Adolesc Psychiatry. 2011 Nov;20(4):315-24.

approve escitalopram for treatment of adolescent depression was "premature, given the available evidence." The decision was unusual in that it was based on a single positive RCT, rather than the usual two. The FDA relied on adolescent data from citalopram MD-18, but FDA's Thomas Laughren testified that if MD-18 were negative, escitalopram should not have been approved for adolescents.[39] In any event, it was inappropriate for the FDA to extrapolate the adolescent data from MD-18 to find efficacy for escitalopram when there were two negative studies of (es)citalopram with worrying levels of AEs.

Overall 94404 was best designed, making it the study most likely to yield clinically meaningful results about (es)citatalopram. With regard to efficacy, this best available study of (es)citalopram indicates inefficacy. For the remaining three inferior studies, none demonstrate clinically significant advantage; one reaches statistical significance, a second (when properly analysed) approaches statistical significance, while a third did not outperform placebo.

With regard to harms, the study closest to real-life condition shows disturbing levels of psychiatric AES and self-harm in the (es)citalopram group. The other studies, when data are available in the CSR are properly analysed, are all consistent with harmful effects from the drug. It is likely that access to individual patient level data would reveal a harm profile that is more troubling still.[40]

[39] Laughren deposition, p401-402
[40] Le Noury J et al. Restoring Study 329: efficacy and harms of paroxetine and imipramine in treatment of major depression in adolescence. *BMJ*. 2015 Sep 16;351:h4320.

Should this case go to trial, I anticipate testifying about documents and matters set out herein, as well as matters discussed in the various declarations I have submitted in relation to this litigation.[41]

Please let me know if there are any further issues that require consideration.

Yours sincerely

Prof Jon Jureidini, MB BS, PhD
Senior Consultant Child Psychiatrist
Research Leader, CEMH
University of Adelaide

[41] My medico-legal work is carried out in my capacity as Professorial Fellow at the University of Adelaide. I am a part-time employee of the University of Adelaide, but receive no direct income from medico-legal work. For testimony, the fee is $US3200/day for up to 8 hours, plus $250/hr (up to a maximum of $2000/day) for extended travelling time.

Appendix 1: Summary of studies

Study	N		Duration	Dose	Age	Criteria	Suicide exclusion	Primary outcome	SAE		Withdrawn	
Citalopram	C	P							C	P	C	P
94404 (von Knorring, 2006)	121	112	12 weeks	26mg	13-18	BDI score was ≥21 for girls and ≥16 for boys GAF score was ≤60 for any of the 4 items assessed	None	change from baseline on the Kiddie-SADS-P	22 18%	17* 15%	42 34%	38 35%
18 (Wagner, 2004)	89	85	8 weeks	24mg	7-17	minimum score of 40 on CDRS-R	Patients who were considered a suicide risk (active suicidal ideations), who had made a serious suicide attempt within the past year, or who had ever been hospitalized because of a suicide attempt	change from baseline in CDRS-R score at Week 8.	0	1 1.2%	18 20%	18 21%
Escitalopram	E	P							E	P	E	P
32 (Emslie, 2009)	155	157	8 weeks	13mg	12-17	45 or greater on the CDRS-R CGI-S score of 4 or greater	Patients who were considered to be a suicide risk (active suicidal ideation), who had made a suicide attempt, or who had ever been hospitalized because of a suicide attempt	change from baseline to Week 8 in the CDRS-R total score	4^ 2.6%	1 0.6%	29 18.7%	24 15.3%
15 (Wagner, 2006)	131	133	8 weeks	12mg	6-17	minimum score of 40 on CDRS-R	Patients who were considered a suicide risk (active suicidal ideation), who have made a serious suicide attempt within the past year, or who have ever been hospitalized because of a suicide attempt.	change from Baseline to Week 8 in CDRS-R	3 2.3%	2# 1.5%	29 22%	18 14%

*CSR claims Patients with SAEs: P= 16 14.3%; C= 18 14.9%

^ CSR says 4/2. Should be 5/1 if 0323202 who made suicide attempt after being treated with escitalopram after withdrawal from study – no suicidal thinking on placebo for 45 days

#CSR claims 2/3, but again SAE occurred post withdrawal on escitalopram

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Appendix 2: JON JUREIDINI RELIANCE LIST

eCTD for Lexapro (adolescent indication)

Documents in support of MSJ (SOL)

1. Kenneth J. Rothman's, *Epidemiology: An Introduction* (2002) excerpts.
2. *Publication and Disclosure Issues in Antidepressant Pediatric Clinical Trials before House Subcomm. on Oversight and Investigations of the Comm. on Energy and Commerce*, 108 Cong., Serial No. 108-121 (Sept. 9, 2004) excerpt.
3. Excerpts of the deposition of Lawrence S. Olanoff, M.D., Ph. D., taken on October 24, 2016.
4. Irving Kirsch et al., *The Emperor's New Drugs: An Analysis of Antidepressant Medication Data Submitted to the U.S. Food and Drug Administration,* 5 PREVENTION & TREATMENT 23, 1-11 (2002).
5. Irving Kirsch et al., *Initial severity and antidepressant benefits: A meta-analysis of data submitted to the Food and Drug Administration*, 5 PLoS Med. 2, 260-68 (2008).
6. Jay C. Fournier, et al., *Antidepressant Drug Effect and Depression Severity: A Patient-Level Meta-analysis*, 303 J. Am. Med. Assoc. 47-53, 47 (2010).
7. Kefauver Harris Amendment, Pub. L. No. 87-781, 76 Stat. 780 (1962).
8. Declaration of James I. Hudson, M.D., SC.D. in this litigation, dated April 5, 2016.
9. Forest's Statement of Undisputed Facts, dated October 25, 2013, in *Wilcox v. Forest Laboratories, Inc.*, 10–CV–10154 (D. Mass.).
10. Excerpts of the deposition of William E. Heydorn, Ph. D., taken on October 14, 2016.
11. Excerpts of the deposition of Charles Flicker, Ph. D., taken on November 4, 2016.
12. Excerpts of the deposition testimony of Lawrence Olanoff, taken on July 18, 2007, in *In re Forest Laboratories, Inc. Securities Litigation*, 05-CV-2827 (S.D. N.Y.).
13. Excerpts of the deposition of Steven L. Closter, taken on October 6, 2016.
14. Final Study 94404 report, "*A double-blind study comparing citalopram tablets (Lu 10-171, 10-40 mg per day) and placebo in the treatment of major depression in adolescents*," dated March 21, 2002.
15. Excerpts of the Final Study Report for CIT-MD-18, entitled "*A Randomized, Double-Blind, Placebo-Controlled Evaluation of the Safety and Efficacy of Citalopram in Children and Adolescents with Depression*," dated April 8, 2002.
16. Excerpts of the Study Protocol for CIT-MD-18, entitled "*A Randomized, Double-Blind, Placebo-Controlled Evaluation of the Safety and Efficacy of Citalopram in Children and Adolescents with Depression*," dated September 1, 1999.
17. Email from Joan Barton, Bates numbered MDL-FORP0168046-0168047, listing investigational sites where patients were unblinded in the CIT-MD-18 study.
18. Defendant's Responses and Objections to Plaintiffs' First Set of Requests for Admission (Revised), dated September 27, 2016.
19. Draft letter by Paul Tiseo to investigators regarding unblinding, dated March 2, 2000.
20. Facsimile sent by Paul Tiseo to investigators of MD-18, dated March 2, 2000.
21. Excerpts of the deposition of James Jin, Ph. D., taken on October 21, 2016.
22. PharmaNet conference notes, dated October 4, 2001.
23. Email from biostatistician Jane Wu regarding results of the CIT-18, dated August 10, 2001.
24. Email from Amy Rubin editing letter to FDA regarding results of the CIT-18, dated March 15, 2000.
25. Letter from Tracy Varner to the FDA regarding a packaging error, dated March 20, 2000.
26. Forest internal emails concerning the timeline of the publication of CIT-MD-18 and their intention of assigning said manuscript to Dr. Wagner as the author.
27. Email from Mary Prescott to Jeffrey Lawrence discussing the intention of Forest to have the Karen Wagner CIT-MD-18 manuscript ghostwritten by Weber Shandwick.
28. Email exchanges between Christina Goetjen and Nefertiti Green regarding promoting the MD-18 data in marketing and public relations activities.
29. Email correspondence between Natasha Mitchner, and William Heydorn containing the Karen Wagner posters.
30. Email correspondence between Natasha Mitchner, and Christina Goetjen containing the Karen Wagner abstract with the latest edition of the Pediatric Data as it was submitted to the American College of Neuropsychopharmacology (ACNP).

31. Forest internal emails attaching the Wagner final slides as submitted to ACNP.
32. Excerpts of the deposition of Natasha A. Mitchner, taken on December 11, 2015.
33. Email correspondence between Lawrence Olanoff, and William Heydorn regarding negative results of the study 94404 not being included in the ACNP posters.
34. Forest selection call notes regarding Wagner data presented to physicians.
35. Forest's press release dated December 13, 2001.
36. Forest internal emails regarding pediatric press release and its false claims of efficacy in children.
37. Forest Pharmaceuticals, Inc., *A Closer Look at Identifying Depression in Children and Adolescents*, dated March 11, 2002.
38. Forest selection call notes regarding Wagner presentation for the treatment of pediatric depression.
39. Forest Pharmaceuticals, Inc., FY'04 Lexapro Strategic Operations, dated Dec. 3, 2002. This document was produced through the course of discovery.
40. Excerpts of the deposition of William E. Heydorn, Ph. D., taken on August 29, 2007, in *In re Forest Laboratories, Inc. Securities Litigation*, 05-CV-2827 (S.D. N.Y.).
41. Email exchanges between Kerstin Fredricson Overo and William Heydorn regarding publication of Study 94404.
42. Forest Laboratories' article published in the American Journal of Psychiatry in June 2004, entitled "*A Randomized, Placebo-Controlled Trial of Citalopram for the Treatment of Major Depression in Children and Adolescents.*"
43. New York Times article, "*Medicine's Data Gap – Journals in a Quandry; A Medical Journal Quandary: How to Report of Drug Trials*" dated June 21, 2004.
44. Forest's press release dated June 24, 2004.
45. Charles E. Grassley's letter to Forest, dated August 3, 2004, requesting more information on the 2002 study of Celexa and its use in children and adolescents.
46. Notice of Intervention, *United States ex rel. Gobble v. Forest Labs., Inc*., No. 05-CV-10201 (NMG) (Dkt. 57) (D. Mass.).
47. Order Unsealing Case, *United States ex rel. Gobble v. Forest Labs., Inc*., No. 05-CV-10201 (NMG) (Dkt. 64) (D. Mass.).
48. Gobble docket, *United States ex rel. Gobble v. Forest Labs., Inc*., No. 05-CV-10201 (NMG) (Dkt. 1) (D. Mass.).
49. Criminal plea agreement, dated September 15, 2010 in *United States v. Forest Pharmaceuticals, Inc.,* 10-CR-10294-NG (D. Mass).
50. Civil Settlement Agreement and Release.
51. Transcripts of an Arraignment on Information, wherein Forest Pharmaceuticals pleaded guilty to three Counts of violations of the Food, Drug, and Cosmetic Act, dated November 19, 2010 in *United States v. Forest Pharmaceuticals, Inc.,* 10-CR-10294-NG (D. Mass).
52. Authorization for Herschel S. Weinstein, Esq. to enter plea, dated September 14, 2010.
53. Defendant's Responses and Objections to Plaintiffs' First Set of Requests for Admission and Third Set of Interrogatories, dated October 11, 2013, *Luster v. Forest Pharmaceuticals, Inc.,* 0922-CC08347 (Mo. Cir. Ct.).
54. Forest Laboratories' Celexa FY01 Marketing Plan.
55. Forest Laboratories' Celexa FY02 Marketing Plan.
56. Forest's Strengths, Weaknesses, Opportunities, Threats (SWOT) analysis, dated April 6, 2000.
57. Celexa Weekly Performance Summary Report, dated May 12, 1999.
58. Selection of off-label call notes showing 44 different sales representatives in 27 different regions engaging in off-label promotion of Celexa and Lexapro in children.
59. Celexa call note regarding Dr. Elizabeth Kressley, dated August 23, 2001.
60. Celexa call note regarding Dr. Lucyna Puszkarska, dated October 29, 2001.
61. Celexa call note regarding Dr. Abraham Rodriguez, dated September 28, 2001.
62. Celexa call note regarding Dr. Ronald Davidoff, dated March 16, 2001.
63. Celexa call note regarding Dr. Edgar Jackson, dated August 11, 2003.
64. Memorandum provided by Len Monteleone to one of the field representatives regarding clever ways to encourage physicians to use Celexa and Lexapro in children.
65. Gerard Azzari curriculum vitae.
66. Excerpts of the Deposition of Gerard J. Azzari, taken on July 21, 2016.
67. Excerpts of the Deposition of Terry L. Nelson, taken on April 1, 2016.

Deposition transcripts and their exhibits:
Heydorn (Kiossovski)
Jin (Kiossovski)
Flicker (Kiossovski)
Olanoff (Kiossovski)
Gergel (Painters)
Wagner (In re Celexa)
Laughren (1/27/17)

Literature:
Wagner - A Randomized Controlled Trial of Citalopram for MDD in Children
Wagner - Poster Efficacy of Cital in the Treatment of MDD in Children
Wagner letters to editor
Psychcentral.com - Despite Controversy, Lexapro Approved for Kids
Carandang - A Review of Escitalopram and Citalopram in Child and Adolescent Depression
BMJ - Blinding important in subjective outcomes
Emslie - Escitalopram in the Treatment of Adolescent Depression (Lexapro)
Blease - The duty to be Well-informed The case of depression
Tonkin - Wishful thinking - antidepressant drugs in childhood depression
Lenzer - Why we can't trust clinical guidelines
Healy - Manufacturing Consensus
Abramson - The effect of COI on biomedical research and clinical practice guidelines
Cosgrove - Conflicts of Interest and Disclosure in the APA's Clinical Practice Guidelines
Le Noury - Restoring Study 329-efficacy and harms of Paroxetine & Imipramine
Goodman - SSRI ads questioned
Leo Lacasse - Media and chemical imbalance
Lacasse - Serotonin & Depression
Pies - Psychiatry's New Brain-Mind and the Legend of the Chemical Imbalance
Jureidini Amsterdam Leemon CIT-18 Int J Risk & Safety
Jureidini Amsterdam McHenry 2016
Jureidini - Efficacy & Safety of Antidepressants for Children - Version 1
Tonkin - Wishful thinking - antidepressant drugs in childhood depression
Jureidini McHenry – KOLs & Overprescribing
Laughren - The scientific and ethical basis for placebo-controlled trials in depression and schizophrenia-FDA perspective

Miscellaneous:
MDL-FOREM0001014 - Email re Peds Manuscript 4-17-02
MDL-FOREM0000904 001.Wagner Hot Topic3.Ppt [Read-Only]
MDL-FOREM0001515 - Email re Ped Data 10-15-01
MDL-FOREM0000973 - Email re Peds Manuscript 1-2-02
MDL-FOREM0000918 - Email re Ped Data 10-16-01 (2)
MDL-FORP0030656 - Publications Timeline
MDL-FOREM0021245 - Emslie Peer Review + manuscript edits
MDL-FOREM0021245 - Emslie Reviewer Comments
MDL-FOREM0021245 - Emslie Reviewer Comments
MDL-FORP0020561 - Varner letter to Katz [3-20-2000]
MDL-FORP0175697 - Urgent message
MDL-FOREM0010758 - List Of Pediatric Studies1 (House) & Grassley Draft Letter
MDL-FORP0020561 (2)
MDL-FORP0023960
MDL-FORP0175697
MDL-FORP0179246 - Presentation about Dif. of Stat v. Clinic. Significance

Letter 7-16-2001 Re Study Results 94404
Email 7-16-2001 Re 94404 Headline Results
Forest Support for Wagner
Celexa Lexapro Hudson Declaration
CIT-MD-18-CRF 507

Simpson to Korotzer re MD-15 and 94404
Nov 19, 1990 ad comm excerpt of transcript
FDA Guidance - blinding highlights
Simpson to Korotzer re MD-15 and 94404
Table 4-1B

Appendix 3 CURRICULUM VITAE for Jonathan Norman JUREIDINI

DOB	2 July 1956
Citizenship	Australia and United Kingdom

Current academic status

Health Sciences
Head, Paediatric Mental Health Training Unit (PMHTU), University of Adelaide
Professor, Disciplines of Psychiatry and Paediatrics, University of Adelaide
Research Leader, Critical and Ethical Mental Health research group, Robinson Research Institute, University of Adelaide
Humanities
Senior Research Fellow, Department of Philosophy, Flinders University

Current clinical position

Senior Child Psychiatrist, Women's and Children's Hospital, Adelaide

Education, professional training and qualifications

Academic
1980 MB BS, University of Adelaide (Passed final exam with Distinction)
1998 PhD, Philosophy Department, Flinders University

Professional
1986 FRANZCP
1987 Accredited Child Psychiatrist, RANZCP
(Specialty registration in psychiatry, Medical Board of SA, Registration number 8441)

Postgraduate Scholarships/Awards
1986 South Australian Health Commission Scholarship for Specialist Study Overseas
1999 Robert J Stoller Foundation Prize for a post-doctoral author
2006 Margaret Tobin Award for "excellence in the provision of services to people with a mental illness who are most in need or most at risk"

Previous roles (since gaining FRANZCP)

1986–1987	Fellow in Child Psychiatry, New South Wales Institute of Psychiatry
1987	Clinical Associate, Young People's Unit, Edinburgh
1987–1988	Clinical Associate, Adolescent Department, Tavistock Clinic, London
1988	Research Fellow, Royal Free Hospital Medical School, London
1988–1994	Director, Adolescent Psychiatry, Women's and Children's Hospital
1993	Visiting Scientist, Adolescent Department, Tavistock Clinic, London
1995–2000	Director, SA Child & Adolescent Training Programme
2007	Visiting Scientist, Therapeutics Initiative, University of British Columbia, Vancouver
1994-2012	Head, Department of Psychological Medicine, WCH

Teaching roles

1995–2000	Director, SA Child & Adolescent Psychiatry Training Programme
2004–	PhD supervision
2010 & 2011	Professor Derek Frewin Citation for Clinical Teaching

Offices in professional and community bodies

Current
2003– Siblings Australia (Chair, 2008-2012)

2009–	Guidelines Working Party, Ethics Expert Advisory Committee, RACP
2010–	Chair, Australian-Palestinian Partnership for Education and Health
2013-	International Centre for Health Communication
Previous	
1993–1997	SA Aboriginal Health Council Research Ethics Committee
1998–2000	President, Psychotherapy Association of South Australia
2002–2004	Management Committee, Justice for Refugees SA
1996–2005	Bi-national Executive, RANZCP Section of C-L Psychiatry
2004–2008	Assessor, Administrative and Disciplinary Division, District Court
2002–2009	Chair, Healthy Skepticism
2011–2012	NHMRC ADHD Expert Working Group
2006–2013	WCH Drug and Therapeutics Committee
2011–2013	South Australian Medicines Advisory Committee
1992–2013	WCH Patient Care Ethics Committee
2011–2015	SA Child Health Clinical Network Steering Committee
2012–2015	SA Formulary Committee

Research Grants as Chief/Partner Investigator

2016-2017

1. **Financial Markets Foundation for Children**
   Jureidini J, Raven M, Tonkin A.
   Antipsychotic prescribing and use among Australian children and adolescents
   $92,323.04

2015-2017

2. **ARC Linkage Grant** (LP140100563)
   Della P, Slade D, Dhaliwal S, Dunston R, Walsh J, Jureidini J, et al
   Driving health care efficiencies and patient care outcomes by improving communication in acute to primary transitions of care
   $480,000

2013-2014

3. **Channel 7 Children's Research Foundation Grant**
   Roberts R, Strohm K, Jureidini J, Giallo R, Robb J.
   Evaluation of a group program for siblings.
   $70,000

2011-2014

4. **ARC Linkage grant** (LP110100035)
   Slade D, Manias E, Battersby M, Scheeres H, Della P, Jureidini J et al.
   Effective clinical handover communication: improving patient safety, experiences and outcomes.
   2011: $308,642, 2012: $141,605, 2013: $267,998.

2000–2003

5. **ARC Large Grant** (A726)
   Jureidini J.
   Narrative and Psychopathology: the role played by narrative in understanding patterns of development and breakdown in psycho–social functioning.
   $80,000

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Reviewer

- Research grant applications, including NH&MRC.
- International journals, including *JAMA, BMJ, Pediatrics, PLoS, Lancet.*