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occurred in a patient who had had multiple suicide attempts in the past, and who had been on modafinil for at least 4 years. The acute delirium occurred in a 19 year old man who was discontinued from treatment after 3 months. The description of the event is unclear in the submission, but the sponsor felt assessment of the event was difficult because of "psychic disturbances and the use of therapeutic sedatives". There is no further information about the case of visual disturbance, because it occurred in a compassionate use program in Canada, in which no CRFs were kept.

SERIOUS ADVERSE EVENTS

A total of 27 patients in extensions of Studies 301 and 302 experienced serious adverse events who were not reported in the original NDA. As Dr. Freiman describes, none of these SAEs can be clearly related to treatment with modafinil.

Approximately 12 patients in Lafon sponsored studies had serious adverse events who were not reported previously in the NDA. Reports of interest included generalized epileptic attack and epileptic attack. No further information about these events is given, other than that Lafon tabular information summaries were used to generate the table (Sponsor's Table 28, Vol 19, page IV-80 of the 6/30/98 submission) in which these events are included).

OTHER ADVERSE EVENTS

The only additional adverse event of interest relates to events subsumed under the COSTART term Dyskinesias. While this term is included in the label in the 1% Table of AEs in the RCTs (2% vs 0% in pbo patients), we have only recently (submission dated 12/14/98) received detailed information about the events coded as dyskinesias.

According to the sponsor, there were a total of 21 modafinil treated patients who experienced a total of 26 events coded as dyskinesia; 12 (2%) of these subjects were in the Phase 3 controlled trials (or extensions), and 9 (1%) were in the foreign, non-Cephalon studies. Two of the events (both in the same patient) were considered serious; a 15 year old young woman who ingested an accidental overdose (1200 mg) and experienced "motoric crawling" and "abrupt movements".

Most of the investigator terms which were subsumed under the term Dyskinesia involved bucco-facial movements (e.g., facial tic, involuntary tongue movements, repetitive tongue rolling, jaw clenching, chewing lower lip, bruxism, rolling tongue).

Five patients discontinued secondary to these events.

The mean time from initiation of treatment to onset of symptoms was 100 days in the Phase 3 trials, and the mean duration of symptoms was 111 days. Of the 11 events in the foreign database, all, save 1, occurred in studies of patients with mood and psychosocial disorders. The 1 other event occurred in a PK study.

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The sponsor states that 18 of the AEs resolved without residual (I cannot tell if the drug was discontinued in some proportion of these cases) and 8 events were on-going, "at the time of discontinuation". There is insufficient information to know how long after discontinuation, if at all, any of these 8 events persisted. Of the 15 events of dyskinesia in 12 patients in the Phase 3 studies, 11 events (10 patients) spontaneously resolved with continued treatment, although 1 patient had involuntary tongue movements that lasted for 1 month, spontaneously resolved, but then recurred 2 months later, and persisted for an additional 1 ½ months. The other events were persistent, but treatment was continued.

Although Dyskinesia is listed in the Adverse Event table, I believe that additional explanation of the type of dyskinesia seen is worthwhile, given that most prescribers would not ordinarily understand this term to primarily refer to oro-facial abnormal movements (despite the fact that the phenomena noted here appear to resemble those seen in patients with tardive dyskinesia, there are other differences, including the much more rapid onset of these movements compared to TD, and the spontaneous resolution without change in the treatment). I believe an adequate solution would be to place an asterisk on the term Dyskinesia in the table, with a footnote stating *Oro-facial abnormal movements.

FOREIGN LABORATORY DATA

As Dr. Freiman notes in his review of 12/15/98, no significant laboratory abnormalities were noted in these patients

APPEARS THIS WAY ON ORIGINAL

Pharmacology

[redacted]

Biopharmaceutics

[redacted]

Labeling

The sponsor proposed extensive changes to the labeling accompanying the Approvable letter. Division staff has had extensive discussions about labeling with the sponsor in several telephone conferences, which included staff from biopharmaceutics and, most notably, Dr. Klein of HFD-170, who reviewed the drug abuse data. The most recent

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version of labeling was submitted to the Division in a fax dated 12/14/98. This version of labeling differs from the version attached to the Approvable letter in some important ways, partly because the sponsor has drafted sections that were relatively incomplete at the Approvable stage. In the following comments, I have highlighted the major changes. It should be noted that the sponsor disagrees vehemently with some of the language included in the labeling we are forwarding with this package; I will highlight which sections are still matters of controversy (we simply have not had sufficient time, given the rapidly approaching due dates, to enter into further negotiations with the sponsor on these points).

Description Section

In our original labeling, we included a statement that Modafinil is chemically unrelated, but pharmacologically related, to other CNS stimulants (i.e., methylphenidate and amphetamine). In reality, modafinil does share some pharmacological actions with the other stimulants, but also differs in some important aspects (striking a balance in describing these similarities and differences has been a major issue in various sections of labeling). In order to avoid a detailed explanation in this section, we have removed the original statement.

Clinical Pharmacology Section

Again, language describing that the effects of modafinil are similar, but not identical, to those of the other stimulants has been added. In addition, language about modafinil's effects at the dopamine receptor has been included, as has language further describing its lack of sympathomimetic activity in various test systems, which is basically a slight expansion of our original wording.

Finally, there is a brief discussion of a study which showed that CNS stimulants in the cat activated a wide array of brain systems, whereas modafinil activated discrete brain areas. This is followed by a statement that the relevance of this finding to humans is unknown.

Pharmacokinetics

Extensive changes have been made in the Absorption and Distribution and Metabolism and Elimination subsections. In particular, a more detailed description of the metabolism of modafinil and drug-drug interactions has been included, as has a discussion of the effects of age on the PK of modafinil.

Clinical Trials

Minor changes have been made to this sub-section.

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Precautions

A paragraph on Abuse Potential, which is essentially repeated in the Drug Abuse section, was placed here in the Approvable letter, and is still here. Dr. Klein feels it should be in this position (given the potential for off label uses and his feeling that the drug intrinsically has considerable abuse potential; recall that his group originally proposed that the drug be Category III under the CSA), but the sponsor strongly objects to its placement here because similar language appears in the Drug Abuse and Dependence section, and labeling for other Schedule IV drugs does not include such language in the Precautions Section.

Drug Interactions

Extensive changes have been made to this section, particularly in the sub-section entitled *Potential Interactions with Drugs That Inhibit, Induce, or are Metabolized by Cytochrome P-450 Isoenzymes and Other Hepatic Enzymes.*

Adverse Events

In our original label, we included a statement about a patient who experienced heart arrest, and we asked the sponsor for additional information. The sponsor submitted more details of this patient, who apparently had a history of syncopal episodes, and a subsequent episode of pain induced bradycardia. However, the episode of arrest referred to in labeling (a 9 second episode of asystole), was unaccompanied by any obvious stimulus (the patient was at the time being observed in a sleep lab). The sponsor has proposed language in this section that implies that the event is not drug related, because of the patient's history.

My view is that there is no evidence linking this event to previous syncopal attacks, and certainly no reason to link this event with the subsequent presumed vaso-vagal event. The language in the version we are sending up includes a description of the specific event and the fact that the patient had previous syncopal attacks. It implies nothing about the cause of this specific event.

Drug Abuse and Dependence

Extensive changes have been made here. Most of these changes are not substantive, and consist of some re-wording of statements in the Approvable draft.

However, the sponsor has proposed the inclusion of a brief description of 2 studies. In one, subjective effects were evaluate in normals who received a single dose of 300 mg modafinil, 300 mg of caffeine, and 15 mg of amphetamine. The sponsor concludes that modafinil was differentiated from amphetamine, but not from caffeine.

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The second study evaluated the abuse potential of 3 doses of modafinil compared to that of 2 doses of methylphenidate in in-patients experienced with drugs of abuse. The sponsor concludes that modafinil produced psychoactive and euphoric effects and feelings consistent with other CNS stimulants (methylphenidate). The section describing these 2 studies is followed by a statement that studies of these kinds are not known to reliably predict the abuse potential of these drugs.

Dr. Klein feels very strongly that the description of the first study should not be included in labeling (see the attachment to his e-mail of 12/10/98). His conclusion is based on the following: 1) the use of a normal, not drug experienced, sample is an inappropriate sample in which to assess the abuse potential, 2) only a single dose was studied, 3) the dose was relatively low, 4) the response to caffeine is dependent upon the level of the tolerance of the subjects to it, which was unknown, 5) the methodology is inappropriate to assess craving, another stated objective of the study. For these reasons, he feels that the conclusions reached by the sponsor are wrong, and therefore that they should not be included in labeling because they are misleading. He does feel that the description of the second study should be in labeling.

Of course, the sponsor wants the description of the first study in labeling, ostensibly because they believe it says something meaningful about the abuse potential in normals, and it provides "balance". My inclination is to agree with our expert consultant, Dr. Klein.

Dosage and Administration

Our original labeling stated the dose as 200 mg/day, given as a single dose in the morning. In a note to the sponsor, we informed them that an additional statement, describing that a single 400 mg/day dose was tolerated, but that there is no evidence to suggest that it confers additional benefit beyond that of the 200 mg dose, might be acceptable, if the requested safety information bore this out.

I believe that such a statement is warranted, based on the sponsor's submission. However, the sponsor requests that this section describe the 200 mg dose as the recommended starting dose, and that there is a suggestion that a daily dose of 400 mg might provide an additional benefit for some patients, and that:

"Accordingly, whether or not to employ a dose of 400 mg is a matter of prescriber and patient preference."

The sponsor asserts that their proposed language is similar to the language in the Aricept label, which they believe is an appropriate comparison based on the Aricept data.

The estimate of the treatment effect on one of the primary outcomes in the first study is slightly numerically greater for the 400 mg dose compared to the 200 mg dose, but in the second study this ordering is reversed. For the proportion of patients improved on the global (the other outcome for which labeling displays estimates of treatment effect), there

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was numerical superiority of the 400 mg dose compared to the 200 mg dose (72% vs 64%) in the first study, and essential identity for the second study (60% vs 58%). Examination of the estimates of the treatment effect on other secondary measures revealed no consistent pattern.

In my view, the data are weak for an advantage of the 400 mg dose compared to the 200 mg dose. Inspection of the Aricept labeling suggests that there was consistent numerical superiority of the high dose compared to the low dose. Such a consistent relationship does not exist for the 400 and 200 mg groups in the modafinil studies. I recommend that our proposed language remain; the Clinical Trials section displays the results, and I do not believe the label need further interpret the data for the prescriber in the Dosage and Administration section.

In summary, there are 5 portions of the labeling we wish to see approved with which the sponsor disagrees or which they have not yet seen: 1) the inclusion in the Precautions section of a statement about abuse potential, 2) the absence of a description of a single dose study in normals in the Drug Abuse and Dependence section, 3) the absence of a statement in the Dosage and Administration section that suggests that 400 mg/day may provide a benefit to some patients beyond that conferred by the 200 mg/day dose, 4) the footnote in the Adverse Event table clarifying the type of Dyskinesias that were seen, and 5) the altered description of the case of asystole. As I described above, we have had numerous telephone negotiations over labeling with the sponsor. Given the rapidly approaching PDUFA due date, I do not believe that we have the time, at the divisional level, to further negotiate the language in these sections. I recommend that the application be approved with the label as we have forwarded it, although, of course, we will be available for discussion of these points.

/S/ [redacted]

Russell Katz, M.D.

Cc:
NDA 20-717
HFD-120
HFD-120/Katz/Freiman/Fitzgerald/Atrakchi/Homanny
HFD-170/Klein

ADDENDUM

I have discussed these issues with Dr. Temple, Director, ODE I (conversation of 12/17/98). We agree that the paragraph on Abuse Potential in the Precautions Section maybe removed. The changes described in points 2-5 above will remain

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as I have proposed, with a slight change in the Dosage and Administration, which will now state that there is no consistent evidence of increased benefit of the 400 mg dose compared to the 200 mg dose, and will refer the reader to the Clinical Trials sub-section.

/S/

Russell Katz, M.D.

APPEARS THIS WAY ON ORIGINAL

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December 16, 1998

Cephalon, Inc.

Paul Leber, MD
Director, Division of Neuropharmacological
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CENTER FOR DRUG EVALUATION
AND RESEARCH

DEC 17 1998

RECEIVED HFD-120

NDA No. 20-717
PROVIGIL® (modafinil) Tablets

Response to FDA Request

Dear Dr. Leber:

Reference is made to our pending New Drug Application (NDA) No. 20-717, Provigil® (modafinil) Tablets, for the improvement of wakefulness in patients with excessive daytime sleepiness associated with narcolepsy, dated December 27, 1996 and our submission to the NDA of June 4, 1998 containing dose finding results for the reproductive toxicology studies. Further reference is made to the December 15, 1998 request by Anna Marie Homonnay-Weikel, Project Manager, to provide written commitment, including timing of final reports, for the post-approval conduct of the TG.AC dermal transgenic mouse bioassay and development and reproductive toxicology studies in the rat for general reproductive performance/fertility and teratology.

Cephalon, Inc. will conduct the [redacted] for modafinil as requested in the December 31, 1997 approvable letter. Assuming approval of the protocol by the Carcinogenicity Assessment Committee (including doses and vehicle for the positive control) within the first quarter of 1999, Cephalon will initiate the trial in the second the quarter of 1999 and submit the final report to the Agency in the second quarter of 2000.

Cephalon will initiate the rat general reproductive performance/fertility and teratology studies, conducted in compliance with GLP regulations, in the first quarter of 1999. Final reports will be submitted to FDA in the third quarter of 1999.

If you have any questions, please contact me at (610) 738-6531.

Sincerely,

Kenneth L. White, PharmD.
Vice President, Regulatory Affairs

KLW/rp
cc: Anna Marie Homonnay-Weikel

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Cephalon, Inc.

October 30, 1998

Paul Leber, M.D.
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NDA 20-717
Provigil (modafinil) Tablets

NEW CORRESP

Dear Dr. Leber:

Reference is made to our pending New Drug Application (NDA) 20-717, Provigil® (modafinil) tablets, for the improvement of wakefulness in patients with excessive daytime sleepiness associated with narcolepsy, which was dated December 27, 1996.

Further reference is made to the approvable letter dated December 29, 1997 and to our subsequent meeting on September 23, 1998 to discuss metabolic drug-drug interaction studies with modafinil.

The purpose of this submission is to forward minutes of the September 23, 1998 meeting, along with two articles and a final study report requested by Dr. Yuan, "Evaluation of the Inhibition of Cytochrome P450 2C19 by Modafinil, Modafinil Sulfone and Modafinil Acid in Human Liver Microsomes." A Table of Contents is attached for your reference.

If you have any questions regarding this information, please contact me at 610-738-6531.

Sincerely,

Kenneth L. White, Pharm.D.
Vice President
Regulatory Affairs

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FDA Meeting Report
Modafinil
Division of Neuropharmacological Drug Products
September 23, 1998

The purpose of the September 23rd meeting was to discuss Cephalon's plans for metabolic drug-drug interaction studies with modafinil. The meeting is a condition of the approvable letter of December 30, 1997, for PROVIGIL®.

In attendance:

FDA
Anna Marie Homonnay-Weikel
Chandra Sahajwalla Pharmacokinetics
Rae Yuan, Pharmacokinetics

Cephalon
James Cappola, Medical Affairs
Richard Civil, Clinical Research
George McCormick, Drug Safety and Disposition
Philmore Robertson, Pharmacokinetics
Kenneth L. White, Regulatory Affairs

Dr. Robertson presented an overview of information relevant to metabolic drug-drug interactions of modafinil that had been obtained since the proposal was submitted (March, 1998). The items were:

1) Anselmino *et al.* Effect of Modafinil, an Antinarcoleptic Drug, and its Metabolites on Human CYP 2C. Abstract and poster presented at the 12th International Symposium on Microsomes and Drug Oxidations, 1998
2) Grözinger *et al.* Interaction of Modafinil and Comipramine as Comedication in a Narcoleptic Patient. Clinical Neuropharmacology, vol. 21, no. 2, pp. 127 – 129, 1998.
3) Report DM-98-009. Evaluation of the Inhibition of Cytochrome P450 2C19 by Modafinil, Modafinil Sulfone and Modafinil Acid in Human Liver Microsomes
4) Report C1538a/112/PK/UK, "A Balanced Latin Square, Randomized, Crossover Study to Investigate the Single-Dose Pharmacokinetics of Modafinil 200 mg and Dexamphetamine 10 mg When Given Either Alone or in Combination in Healthy Male Volunteers" (Clinical/Pharmacokinetic Report submitted to IND [redacted] Serial No. 096, March 3, 1998 and Clinical Study Report Synopsis submitted September 24, 1998, [redacted] Serial No. 112 – IND Annual Report).

Photocopies of the abstract and poster and of the publication were given to the reviewers during the meeting. This information will be submitted to the NDA in reference to this meeting. Dr. Yuan expressed interest in the effect of inhibition of CYP2C19 by the sulfone metabolite of the drug. Dr. Yuan asked that we submit the recently completed *in vitro* work with the sulfone and this enzyme so that she would be able to review it while final labeling of the drug is being set. A preliminary report would suffice. We agreed to

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supply a report as requested. As noted above, the results of the modafinil-dextroamphetamine interaction study had already been provided. We were also asked whether we planned other inhibition studies with the sulfone and answered that these studies were underway at the present time, but no results are yet available.

In subsequent discussion, the following topics were addressed. They are organized by study, not by their chronological order in the discussion.

In vitro interaction study - CYP2C19 inhibition

Both modafinil and modafinil sulfone are planned for inclusion in the *in vitro* interaction study. At steady state for modafinil (400 mg/day) and modafinil sulfone, the total inhibition of CYP2C19 at Tmax is estimated to be as much as 65% to 75% with the contribution of the sulfone estimated to be 10% to 15%.

The selection of diazepam as a standard substrate for CYP2C19 was briefly discussed, with a question being raised as to the relationship of the concentration selected (10 μM) to the K_M of the compound for CYP2C19. The K_M is *ca.* 24 μM (Jung *et al.*, 1997), compared to a K_M of *ca.* 80 μM for CYP3A4. Use of 10 μM should provide sufficient analytical sensitivity while ensuring that we are targeting primarily the CYP2C19-derived metabolism. FDA asked about the concentrations of modafinil to be used. Proposed concentrations were given as 10, 30, 100 and 300 μM, which were acceptable.

In response to questions, Dr. Robertson reviewed the metabolic pathways of clomipramine and fluoxetine. CYP2D6 is responsible for important metabolic pathways for both of these compounds (as it is for most tricyclic antidepressants and selective serotonin reuptake inhibitors), with CYP2C19 generally having a rather smaller role. However, in CYP2D6 poor-metabolizers, CYP2C19-dependent pathways can take on greater prominence. The primary goal of the inclusion of clomipramine, *des*-methylclomipramine and fluoxetine in this study is to determine the relative contributions of CYP2C19 in their metabolism by using liver microsomes from CYP2D6-deficient, -normal and -super-metabolizer subgroups. This is particularly important given the lack of agreement in reports in the literature regarding the contributions of CYP2C19 to the metabolism of these compounds.

The information derived from the *in vitro* study can then be used to enable a decision as to whether additional clinical study is warranted.

Clinical interaction studies – CYP enzyme induction/suppression

In response to a request from the reviewers, Dr. Robertson presented an overview of the results from the *in vitro* induction study (human hepatocytes) recently completed at XenoTech. Most of the information presented had been included with the proposal, but some elements had been only recently received from [redacted] The new information, primarily the pictures of [redacted] derived from [redacted], was presented. The [redacted] results demonstrated that the concentrations of the CYP3A4, CYP1A2 and CYP2B6 enzymes were increased in the microsomes from the modafinil-treated hepatocytes; hence, enzyme induction, not just enzyme activation had occurred. The [redacted] results for CYP2C9 were equivocal. Dr. Yuan asked if CYP3A4 was inhibited by the sulfone. Dr. Robertson answered that, given the

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poor inhibition of CYP3A4 by parent modafinil (Ki ≈ 632 μM) and the greater polarity of the sulfone metabolite, it would not be expected that modafinil sulfone would be an inhibitor of CYP3A4.

Because of the relatively small number of important pharmaceutical products that have CYP1A2 or CYP2B6 as primary metabolic pathways, Cephalon proposed that it would be unnecessary to conduct clinical drug-drug interaction trial testing those pathways. From the discussions that ensued, it is our belief that the proposal was accepted.

Dr. Yuan asked whether CYP2B6 constitutes a primary metabolic pathway for ketoconazole. Dr. Robertson answered that he thought not, but would check. Subsequently Dr. Robertson has been able to ascertain that ketoconazole is not only a potent inhibitor of CYP3A4/5, but is also a substrate primarily for those enzymes (Spatzenegger and Jaeger, 1995). Consistent with that conclusion, concurrent administration of ketoconazole with rifampin, a potent CYP3A4 inducer, caused marked reduction in serum ketoconazole levels (McEvoy (ed.), 1998). Hence, the contribution, if any, of CYP2B6 to the metabolism of ketoconazole would not appear to be a major one.

CYP3A4 Study – The basic design of the study was accepted, and the difficulty inherent in performance of such a study acknowledged. Our rationale (as summarized in the written proposal) for the length of the dosing period with modafinil was briefly presented and accepted as reasonable.

Given the high inter- and intraindividual variability expected for ethinylestradiol, Dr. Yuan suggested that a second, less variable marker substrate (e.g., triazolam or midazolam) also be included. Even taking into account the potential effects of the steroidal contraceptive on the pharmacokinetics of these compounds, use of one of them would seem likely to give a less equivocal result than would the steroidal contraceptives. We agreed and said that we would try to work such a second marker substrate into the protocol.

CYP2C9 – The rationale for the study was briefly discussed and accepted as reasonable. The rationale centers on the fact that there was a decrease in CYP2C9 activity when human hepatocytes were cultured in the presence of high concentrations of modafinil. It is not known whether this rather unusual finding will correlate with any findings *in vivo*; however, due to the low therapeutic index for warfarin, a study was considered to be warranted.

The timecourse of the suppression effect, if it occurs *in vivo*, is not known, and FDA commented that time could then be a problem with this study. Dr. Robertson replied that we recognize this fact and, by analogy to the induction study, have proposed a relatively long period of treatment with modafinil in order to ensure that we have optimized the probability that we can detect even a relatively small change in CYP2C9 activity.

Many of the points raised in the context of the discussion of the CYP3A4 study were, of course, directly applicable also to the CYP2C9 study. However, as noted above, there is no information available regarding CYP suppression by modafinil *in vivo*.

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The FDA reviewers also pointed out that with multiple pathway for metabolism available in the cytochrome system, we should emphasize proper dose to prevent escape metabolism from masking inhibition of a particular pathway. Dr. Robertson replied that, due to the fact that this study will be in normal subjects, we will have to keep the dose of Coumadin® low. However, given the selectivity of CYP2C9 for (*S*)-warfarin and given our intention to use a stereoselective assay to monitor the reaction, the study should provide a definitive result.

The Reviewers present accepted the plans proposed for drug interaction studies and the basic design of the trials.

Cephalon was requested to submit the new data presented during the meeting. Abstracts and articles presented will be attached to the minutes of the meeting and submitted to FDA. The results of the study of modafinil sulfone inhibition of CYP2C19 will be submitted to FDA in October.

Cephalon was asked for the timelines for initiation and completion of the proposed studies.

1. *In vitro* study in human hepatocytes is scheduled to be initiated in December and completed 6 months later
2. The ethinylestradiol study is scheduled to be completed no earlier than 3rd Quarter of 1999. A draft protocol should be available in November.
3. The study of warfarin is scheduled to be completed by 2nd Quarter of 1999. A draft protocol should be available in November.

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MEMORANDUM OF TELECON

DATE: 12/18/98

NDA: 20-717

DRUG: PROVIGIL (modafinil) Tablets

BETWEEN:

Cephalon, Inc.:

Ken White
Earl Henry
Man Miller
Rich Civil
Pliji Robertson
Nancy Kribbs
George McCormick
Wayne Schuck
Paul Kirsch

AND:

FDA:
Rusty Katz, M.D.
Joel Freiman, M.D.
Anna M. Homonnay-Weikel, R.Ph.

SUBJECT: Inform Cephalon of DNDP's final decisions on the aspects of the proposed labeling that were subject to recent discussions between Cephalon, Inc. and DNDP.

DISCUSSION:

- The 'Abuse Potential' subsection will be deleted from the 'Precautions' section.
- The Division decided not to include information about the single dose study in normals in the 'Abuse Potential' section.
- The 'Dosage and Administration' section will remain the same as the 12/29/97 approvable labeling except for new inclusions concerning dosing in certain patient populations.
- The comparisons in the 'Carcinogenicity' and 'Fertility' sections will be based on the 200 mg dose versus the 400 mg dose.

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- Cephalon, Inc. seemed agreeable to these final decisions regarding the proposed labeling.

/S/ [redacted]

Anna M. Homonnay-Weikel, R.Ph.
Project Manager

cc:

Orig IND
Div File
HFD-120/Katz/Freiman
HFD-120/Homonnay

TELECONFERENCE

APPEARS THIS WAY ON ORIGINAL