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Electroencephalograph effects of single doses of *Ginkgo biloba* and *Panax ginseng* in healthy young volunteers

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Abstract

Both *Ginkgo biloba* and *Panax ginseng* exert a number of physiological effects and have been shown to modulate aspects of cognitive performance. Whilst a number of studies have examined ginkgo's effects on electroencephalograph (EEG) recordings, to date, none have investigated the EEG effects of ginseng. In this double-blind, placebo-controlled, balanced crossover experiment, the effects of single doses of *G. biloba* (360 mg GK501), *P. ginseng* (200 mg G115), and an identical placebo, on auditory-evoked potentials, contingent negative variation (CNV), and resting power within the delta, theta, alpha, and beta wavebands, were assessed in 15 healthy volunteers. Each participant was assessed on three separate occasions 4 h after consuming that day's treatment. The order of presentation of the treatments was dictated by a Latin square with 7 days between testing sessions. The results showed that ginseng led to a significant shortening of the latency of the P300 component of the evoked potential. Both ginseng and ginkgo also led to significant reductions in frontal 'eyes closed' theta and beta activity, with additional reduction for ginseng in the alpha waveband. These findings demonstrate for the first time that *P. ginseng* can directly modulate cerebroelectrical activity, and that these effects are more pronounced than those following *G. biloba.*

Keywords: Ginkgo; Ginseng; Electroencephalograph; EEG; Evoked potentials; Power

1. Introduction

Chronic administration of standardised extracts of *Ginkgo biloba* has been reported to ameliorate cognitive deficits associated with ageing (e.g., Allain et al., 1993; Rai et al., 1991), vascular dementia, Alzheimer's disease (e.g., Kanowski et al., 1996; Le Bars et al., 1997), and cerebral insufficiency (e.g., Kleinen and Knipschild, 1992). A number of studies in these populations/groups have demonstrated modulation of electroencephalograph (EEG) activity. Typically, in the resting eyes closed EEG, this has taken the form of either a reduction in theta waveband activity or a simultaneous decrease in theta and increase in alpha waveband activity. Such a pattern of modulation of relative theta/alpha waveband activity has been interpreted both as a 'normalisation' of activity (Itil et al., 1996), and as being indicative of increased vigilance (Geßner et al., 1985). A similar pattern has been reported following chronic administration of ginkgo to sufferers from age-associated cognitive impairment (Geßner et al., 1985; Pidoux et al., 1983), cerebral insufficiency (Hofferberth, 1995; Schulz et al., 1991), and Alzheimer's disease (Hofferberth, 1994). Both acute and chronic administration of 120 mg of a *G. biloba* extract to sufferers from age-associated cognitive impairment have also been reported to shorten P300 latency (Semlitsch et al., 1995).

A linear, dose-dependent increase, in comparison to placebo, in the resting eyes closed alpha activity (occipital recording) of 12 healthy unimpaired adults has also been reported as a consequence of ingestion of three doses (40, 120, 240 mg) of ginkgo extract (Itil et al., 1996). In a further placebo-controlled, acute dose (80 and 160 mg) study involving eight testing sessions spanning 6 h, Luthringer et al. (1995) reported increased relative and

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absolute alpha-1 (8–9.5 Hz) power in frontal regions of the scalp. Relative alpha-2 (10–12.5 Hz) power was also increased frontally, but the absolute power of this band showed a trend towards a decrease in the same region. Following sub-acute treatment (160 mg for 5 days), a concomitant increase in relative and absolute beta waveband power and a decrease in relative theta waveband power were also noted. In this study, both contingent negative variation (CNV) and P300 amplitude were also investigated; but the pattern of modulation, with both significant increases and decreases over the eight testing sessions, is not easy to interpret. These results, from studies involving healthy cohorts, should also be seen in the light of a previous paper (Kunkel, 1993) that reported the results from two separate acute dosage crossover experiments, and which showed a large number of treatment-related effects. However, there was no clear pattern to the results, with all of the three doses of ginkgo (40, 80, and 160 mg Egb 761) and the two fractions of the extract that were used generating markedly different profiles of significant resting EEG waveband modulation.

The evidence with regards the effects of ginseng is less clear cut. There is little methodologically sound evidence for its efficacy either in the enhancement of cognition or in the treatment of pathological conditions (for comprehensive overviews, see Bahrke and Morgan, 2000; Vogler et al., 1999). It is noteworthy that no study to date has investigated any EEG effects of ginseng.

Previous work from our laboratory (Kennedy et al., 2000; 2001a,b, 2002) has suggested that whilst both ginkgo and, even more so, ginseng led to dose-dependent/specific enhancement of memory performance, ginkgo was associated with a linear dose-dependent improvement in the speed of performing attention tasks (Kennedy et al., 2000). In contrast, both the lowest (200 mg) and highest (600 mg) dose of ginseng led to a deterioration in speed across the same tasks (Kennedy et al., 2001a). Similarly, whilst ginkgo improved the speed of performing computerised mental arithmetic tasks, the lowest dose of ginseng led to reduced speed (Scholey and Kennedy, 2002). In a study attempting to replicate the memory and attentional effects of the two extracts seen in these previous studies, the mnemonic effect of 400 mg of ginseng was replicated. However, 360 mg of ginkgo failed to produce a speeding of attentional task performance but did improve memory performance. The same dose also improved the speed or accuracy of both of the serial subtraction tasks (Kennedy et al., 2002).

In light of this pattern of results, it seems timely to investigate the bioelectrical effects of single doses of both *P. Ginseng* and *G. biloba*. The current study therefore comprised a double-blind, counterbalanced, crossover experiment, involving a single cohort of healthy young (<40 years) volunteers. Both evoked potentials and resting EEG recordings were investigated following administration of a placebo and doses of ginkgo (360 mg) and ginseng (200 mg). These doses were chosen on the strength of their modulation of serial subtraction performance and the most striking opposite effects on the speed of performing attention tasks.

2. Materials and methods

2.1. Participants

Ten female and five male volunteers (mean age 26.6 years, range 19–39 years) took part in the study, which was approved by the Joint Ethics Committee of Newcastle and North Tyneside Health Authority. Prior to participation, each volunteer signed an informed consent form and completed a medical health questionnaire. All participants reported that they were in good health and were taking no illicit drugs. Additionally, they were free of any 'over the counter', herbal, or prescribed medications, with the exception, for some female volunteers, of the contraceptive pill. Habitual smokers consuming more than five cigarettes/day were excluded from the study. Of the 15 participants, 3 were light social smokers and they agreed to abstain from smoking on the day of testing. All participants abstained from caffeine-containing products throughout each study day and alcohol for a minimum of 12 h prior to the first testing session of the morning.

2.2. Treatments

On each study day, participants received eight capsules that were of identical appearance. The individual capsules contained either placebo, 60 mg of *G. biloba* extract (GK501, Pharmaton, Lugano, Switzerland), or 100 mg of *P. ginseng* extract (G115 Pharmaton). Depending on the condition to which the participant was allocated on that particular day, the combination of capsules corresponded to a dose of either 0 mg (placebo), 360 mg *G. biloba*, or 200 mg *P. ginseng*.

2.3. Procedure

The study employed a within-subjects, double-blind, placebo-controlled, balanced crossover design with single-dose administration (placebo, 360 mg *G. biloba* or 200 mg *P. ginseng*) of the treatment on the relevant days. Participants attended on three separate occasions and received the treatment in an order dictated by a Latin square. Each day of the study was separated by a 7-day 'washout' period. EEG recording took place 4 h after ingestion of the day's treatment. The running order of the EEG measures was as follows: CNV; auditory-evoked potentials; 'eyes open' wave band analysis; and 'eyes closed' waveband analysis.

2.4. EEG recording

EEG was recorded from disposable silver/silver chloride gel-filled electrodes attached to the scalp with collodion according to the international 10/20 system. Eighteen recording sites (N_Z, F_Z, C_Z, P_Z, F_3, F_4, F_7, F_8, C_3, C_4, T_3, T_4, T_5, T_6, P_3, P_4, O_1, and O_2), all referred to linked mastoids, were utilised. EEG signals were amplified using a Neuroscan Synamps system (Neurosoft, Sterling, VA, USA). Eye movement compensation was derived from nasion-linked mastoid electrodes. Participants were asked to visually fixate on a small red cross displayed on a monitor in an effort to minimise eye movements during all eyes open EEG recordings. Any sections of the EEG recording still contaminated with eye movements, muscular activity, or other artefacts were excluded from the analysis.

Amp settings (filter) during the three recording periods were as follows: CNV—low pass 30 Hz, high pass 0.02 Hz; auditory-evoked potentials—low pass 30 Hz, high pass 0.15 Hz; and power/frequency spectrum—low pass 100 Hz, high pass 0.1 Hz.

At cutoff frequencies, the voltage gain was approximately -6 dB. The slopes are greater than or equal to -12 dB/octave.

Event-related potentials and wave band analyses were conducted using the Neuroscan 4 Workstation programme (Neurosoft).

2.5. Contingent negative variation

CNV was elicited with warning tones presented binaurally through earphones (1 kHz, 20 ms, 60 dB), followed 1.25 s later by binaural imperative tones (650 Hz, 400 ms, 60 dB) to which the participant responded with a dominant hand index finger push button. The magnitude of CNV (in microvolt seconds) was measured from the average of 10 stimuli. Button press reaction times to the imperative tone were also calculated.

2.6. Auditory-evoked potentials

P300 amplitude and latency were elicited with a standard auditory oddball paradigm. Frequent and target tones were presented binaurally through headphones. Participants were instructed to listen for and count 19, 20 or 21 infrequent target tones (650 Hz, 60 dB, 200 ms), which occurred randomly amongst 82–90 frequent nontarget tones (1 kHz, 60 dB, 200 ms). Interstimulus intervals varied randomly between 1250 and 3000 ms. The latency range in which the P300 maximum amplitude and latency were determined was set at 280–375 ms. However, any peaks outside this range were measured manually, with preliminary visual checking of all peaks prior to measurement.

2.7. Power/frequency spectrum

The power/frequency spectrum of the resting EEG was calculated by Fast Fourier transform of the average of 50 two second continuous epochs of resting EEG activity, eyes open and eyes closed, over the frequency range 0.5–26.8 Hz in 0.487 Hz steps. Total power was expressed as μV^2/Hz in the delta (0.5–3.9 Hz), theta (4.3–7.8 Hz), alpha (8.2–14.1 Hz), and beta (14.6–26.8 Hz) frequency bands. Additionally, given the exploratory nature of this study, further analyses were performed on more restricted frequency bands (see below).

Table 1
Mean (untransformed) P300 amplitude and latency and mean 'eyes closed' power in the delta, theta, alpha, and beta wavebands for *G. biloba*, *P. ginseng*, and placebo

EEG measure	Scalp region	Placebo	Ginkgo	Ginseng
P300	Frontal	7.45	7.45	6.84
Amplitude	Left temporal	10.78	9.89	9.69
(μV)	Right temporal	10.57	10.19	9.94
	Parietal	12.87	11.93	11.44
	Occipital	10.13	9.29	9.04
P300	Frontal	339.79	339.92	***335.35***
Latency	Left temporal	348.27	344.6	***334.18*****
(milliseconds)	Right temporal	337.11	340.24	***334.78***
	Parietal	337.62	342.64	***332.43***
	Occipital	345.2	339	***333.23*****
Waveband		Mean power (μV^2/Hz) 'eyes closed'		
Delta	Frontal	55.09	39.69	48.65
(0.5–3.9 Hz)	Left temporal	14.37	11.44	12.91
	Right temporal	13.32	11.93	17.08
	Parietal	15.52	15.36	14.42
	Occipital	12.66	11.33	11.58
Theta	Frontal	4.46	3.35**	***3.1*****
(4.3–7.8 Hz)	Left temporal	3.28	3.11	***2.87***
	Right temporal	3.28	3.30	***2.92***
	Parietal	4.89	4.97	***4.31***
	Occipital	4.00	4.40	***3.89***
Alpha	Frontal	4.04	3.09	3.05*
(8.2–14.1 Hz)	Left temporal	5.33	4.96	4.91
	Right temporal	5.72	6.11	5.54
	Parietal	9.03	8.59	8.60
	Occipital	17.66	20.37	19.79
Beta	Frontal	0.68	0.52**	***0.51*****
(14.6–26.8 Hz)	Left temporal	0.76	0.67	***0.64***
	Right temporal	0.83	0.80	***0.67***
	Parietal	0.96	0.94	***0.87***
	Occipital	1.14	1.23	***1.08***

Electrodes were grouped into frontal, left temporal, right temporal, parietal, and occipital scalp regions. Bold italicised means indicate an overall significant difference (Dunnett's test) between the relevant condition and placebo. ANOVA and post hoc comparisons of the waveband power were undertaken on log-transformed data (not shown).

* $P<0.05$, significance of post hoc comparisons (Bonferroni t) of the relevant active treatment mean against placebo in the cortical region indicated.

** $P<0.01$, significance of post hoc comparisons (Bonferroni t) of the relevant active treatment mean against placebo in the cortical region indicated.

2.8. EEG descriptive statistical mapping

The mean EEG power (obtained from grand means of the participants data following placebo and the relevant treatment) at each frequency interval of 0.487 Hz across the frequency spectrum (0.5–26.8 Hz) and at each electrode site was calculated both individually and grouped into the four standard frequency bands (delta, theta, alpha, and beta). Paired *t* test values for each 0.487 Hz interval and each frequency band (eyes open and eyes closed) were mapped over the surface of the head using the Neuroscan 'Window' programme, which is based on a linear interpolation algorithm linking each individual electrode with its four nearest neighbouring electrodes.

2.9. Statistical analysis

Mean P300 latency and amplitude and mean frequency band power (delta, theta, alpha, and beta) were calculated during each treatment (placebo, ginkgo, and ginseng) across groups of electrodes representing frontal (F_Z, F_3, F_4, F_7, F_8,), left temporal (C_3, T_3, T_5,), right temporal

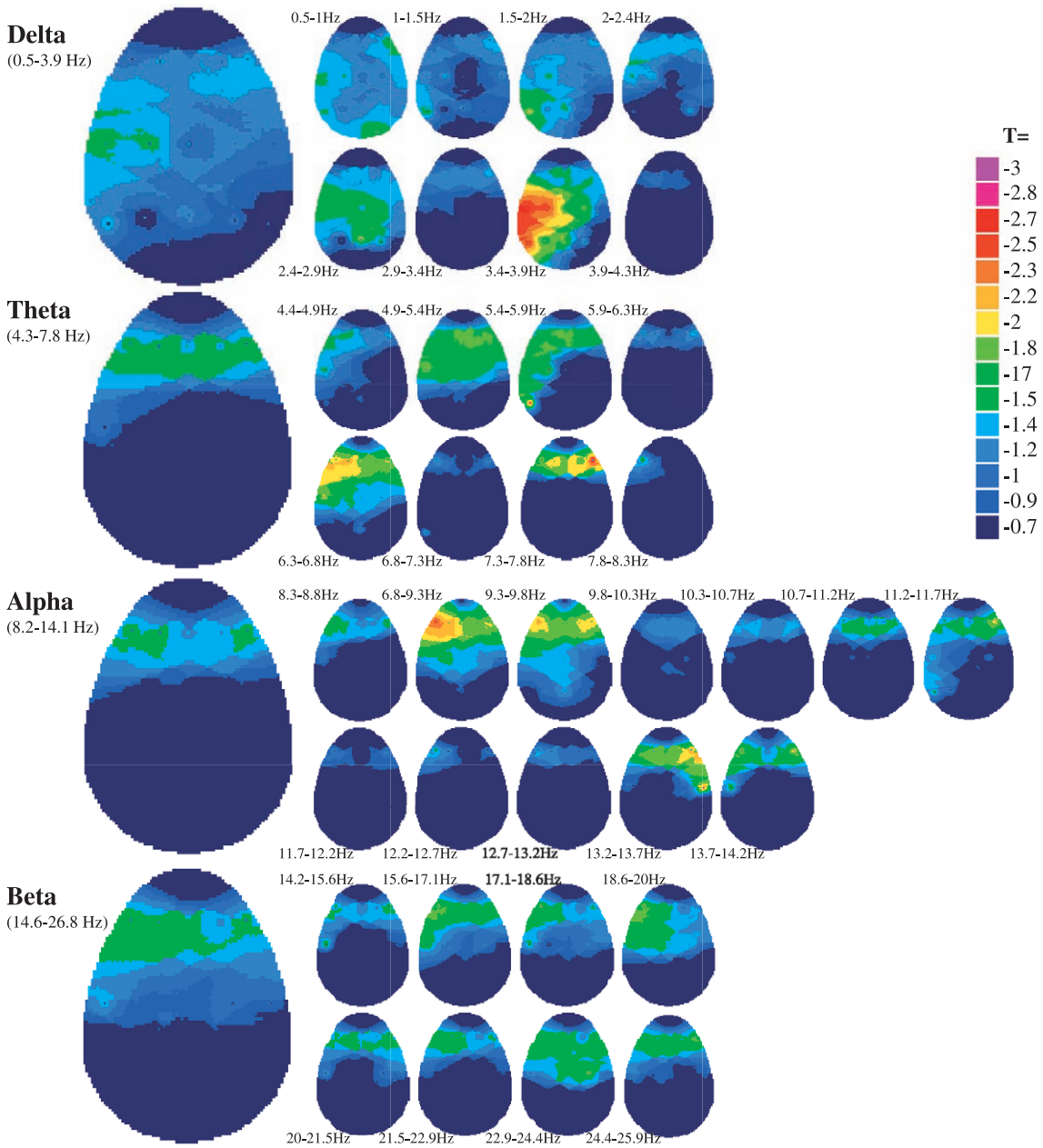

Fig. 1. Descriptive probability maps showing smoothed topographic reductions in EEG power in each waveband, in comparison to placebo, following 360 mg of *G. biloba*. Larger maps represent power averaged across the entire waveband (delta, theta, alpha, and beta), and the smaller maps represent comparisons at the individual 0.487 Hz (1.46 Hz for the wider beta waveband) intervals that make up the larger waveband.

(C_4, T_4, T_6), parietal (C_Z, P_Z, P_3, P_4), and occipital (O_1, O_2) scalp regions. Normality of the data was assessed using the Anderson–Darling test, and measures that deviated from normality were log-transformed to a normal distribution prior to analysis (Gasser et al., 1982). Following any necessary transformation, two-factor (Treatment × Scalp Region) repeated measures analysis of variance (ANOVA) was carried out for each measure. Where appropriate, post hoc comparisons of the individual treatment versus placebo for overall condition means were carried out using Dunnett's test. Individual post hoc comparisons of ginkgo versus placebo and ginseng versus placebo within each individual brain region were made using Bonferroni *t* tests utilising MS error from the ANOVA.

3. Results

Data from each of the four wavebands (delta, theta, alpha, and beta) were log-transformed to a normal distribution prior to ANOVA (Gasser et al., 1982).

P300 amplitude and latency and the mean power (eyes closed) for the frequency bands (delta, theta, alpha, and

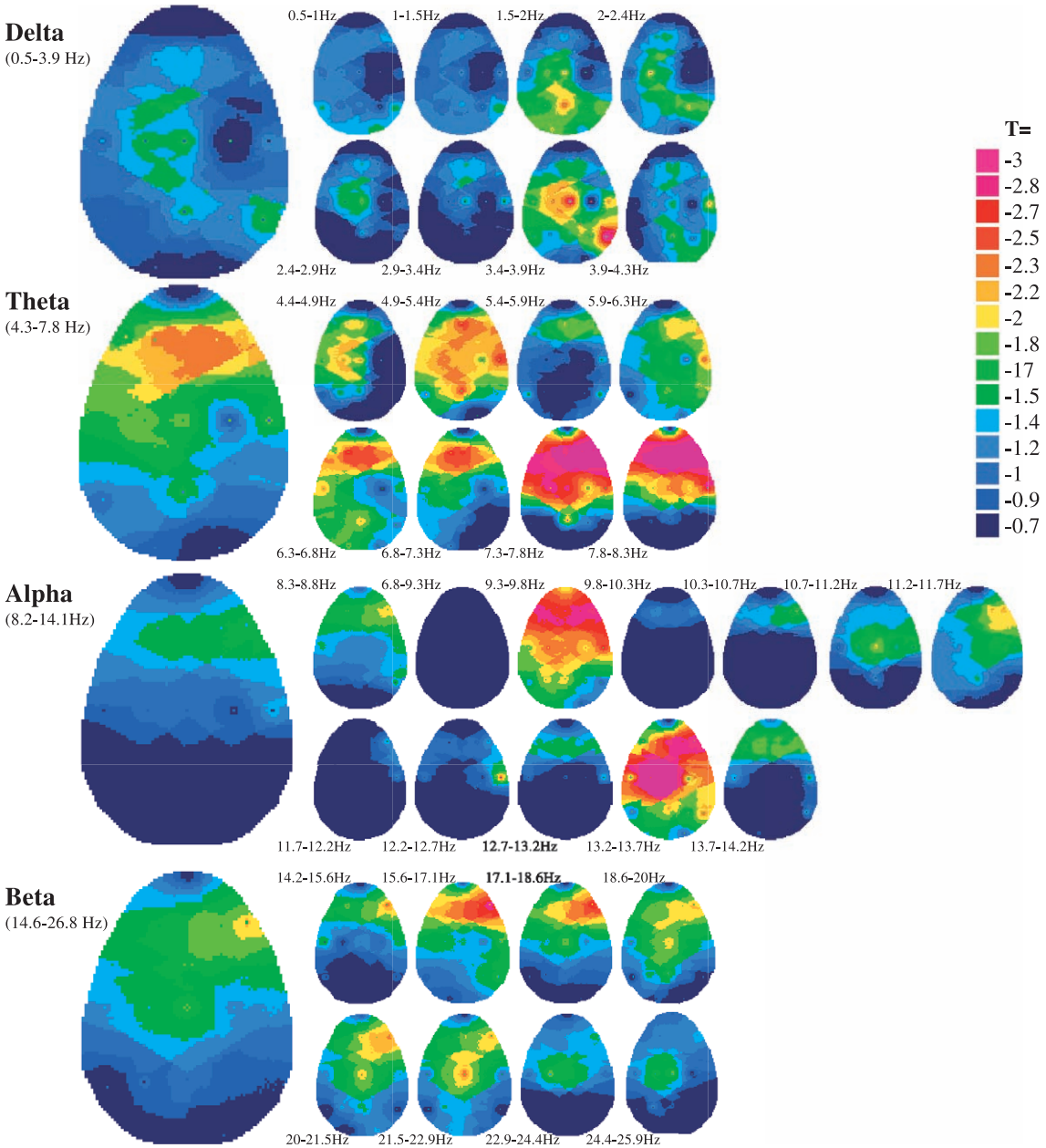

Fig. 2. Descriptive probability maps showing smoothed topographic reductions in EEG power in each waveband, in comparison to placebo, following 200 mg of *P. ginseng*. Larger maps represent power averaged across the entire waveband (delta, theta, alpha, and beta), and the smaller maps represent comparisons at the individual 0.487 Hz (1.46 Hz for the wider beta waveband) intervals that make up the larger waveband.

beta) in the individual brain regions 4 h following ingestion of placebo, 360 mg of *G. biloba*, and 200 mg of *P. ginseng* are presented in Table 1.

3.1. CNV

There were no significant differences on the measures of CNV (data not shown). There were no significant differences in the reaction times to the imperative tones (placebo 166.6 ms, ginseng 160.1 ms, and ginkgo 170 ms).

3.2. P300

Whilst there were no significant differences on the amplitude of the P300 wave, the ANOVA of P300 latency showed that there was a significant interaction between treatment and scalp regions [$F(8,112)=2.07$, $P<.05$). Post hoc comparisons (Dunnett's) showed that the overall latency of the P300 wave was significantly reduced in the ginseng condition ($P<.05$). Comparison (Bonferroni *t*) of each treatment against placebo in the individual scalp regions revealed that ginseng evinced a significant reduction in latency in both the left temporal and occipital groupings of electrodes ($P<.01$ in both cases).

3.3. 'Eyes open' power/frequency wavebands

There were no significant differences on the 'eyes open' waveband analysis (data not shown).

3.4. 'Eyes closed' power/frequency wavebands

3.4.1. Delta (0.5–3.9 Hz)

There were no significant differences on the delta waveband.

3.4.2. Theta (4.3–7.8 Hz)

The ANOVA revealed a trend towards a main effect of treatment on the power of the theta waveband [$F(2,28)=2.93$, $P<.1$] and a significant interaction between the treatment (placebo/ginkgo/ginseng) and the brain regions [$F(8,112)=2.92$, $P<.01$]. Post hoc comparisons (Dunnett's) showed that theta activity was reduced overall by ginseng in comparison to placebo ($P<.05$). Comparison of placebo against each treatment in individual scalp regions (Bonferroni *t*) revealed that power was reduced across the frontal electrodes following both ginkgo and ginseng administration (both $P<.01$).

3.4.3. Alpha (8.2–14.1 Hz)

A significant interaction between treatment and brain regions was evident [$F(8,112)=3.44$, $P<.001$]. Post hoc comparisons (Bonferroni *t*) showed that the power of alpha activity across the frontal scalp region was significantly decreased by ginseng ($P<.05$).

3.4.4. Beta (14.6–26.8 Hz)

A significant interaction between treatment and brain regions was also evident within the beta waveband [$F(8,112)=2.51$, $P<.05$]. Post hoc comparisons (Dunnett's) of treatment means against mean for placebo showed that the power of the beta waveband was reduced overall following ingestion of ginseng ($P<.05$). Power was also specifically reduced in the frontal scalp region following both ginkgo ($P<.01$) and ginseng administration ($P<.01$).

Descriptive topographic probability maps (paired *t* tests at each electrode smoothed with the four adjacent electrodes by a linear algorithm) of each waveband, showing reductions in EEG activity following both treatments in comparison to placebo, are presented in Fig. 1. (ginkgo vs. placebo) and Fig. 2. (ginseng vs. placebo). Both figures include topographic maps showing reductions in activity at every 0.487 Hz interval (1.46 Hz intervals for the wider beta waveband). Alpha was the only waveband that showed any marked (nonsignificant) increase in power.

4. Discussion

The results of this study show that acute doses of *G. biloba* and *P. ginseng* are capable of modulating cerebral bioelectrical activity as measured by EEG. In both cases, acute oral ingestion of the treatment was associated with significant decreases in both theta and beta wavebands, predominantly in frontal scalp areas. Ginseng also resulted in reduced frontal alpha activity. In contrast to our expectations, based on previous demonstrations of faster reaction times for ginkgo and slower responses for ginseng during performance of tasks assessing attention (Kennedy et al., 2000, 2001a), only ginseng was associated with modulation of evoked potentials, with decreased latency for the P300 wave, whilst reaction time data from the CNV test showed no differences in speed for any condition.

Previous research into the EEG effects of *G. biloba* in elderly pathological populations has suggested that the extract's most notable effect is one of reducing the proportion of resting 'eyes closed' theta activity in comparison to alpha activity. Following the ingestion of ginkgo, a reduction in theta activity has been demonstrated in cohorts suffering from Alzheimer's disease and vascular dementia (Hofferberth, 1994) and cerebral insufficiency (Hofferberth, 1995). Similarly, decreases in theta activity with concomitant increases in alpha activity have been shown in patients suffering cerebral insufficiency (Pidoux et al., 1983; Schulz et al., 1991). Other studies have demonstrated similar modulation in subgroups of 'responders' within cohorts suffering from dementia (Itil et al., 1998) and age-related mental deterioration (Geßner et al., 1985). This sampling bias may make the findings of these latter studies less than compelling when considering overall group differences in EEG. Nevertheless, it is of interest to note that the current study found broadly consistent data despite being undertak-

en in a healthy younger cohort. Not only was resting 'eyes closed' theta power significantly reduced across frontal electrodes, but also the relative proportion of theta to alpha activity across the whole scalp was reduced by 3.8% following ginkgo (data not shown).

These data do not, however, support the suggestion that ginkgo increases alpha activity in healthy cohorts (Itil et al., 1996; Luthringer et al., 1995). Indeed, here we found a trend for decreased frontal alpha coupled with increased occipital alpha, both of which failed to reach statistical significance (following log-transformation of the data). It is also noteworthy that whilst Itil et al. (1996) showed a clear ginkgo-associated increase in alpha power in a cohort of 18–65 year olds, there are two reports using topographic EEG following ginkgo in younger cohorts (<40 years), which have found contrasting results. The first found increased frontal alpha 1 (8–9.5 Hz) absolute power, with a concomitant trend towards reduced alpha 2 (10–12.5 Hz) absolute power in the same region (Luthringer et al., 1995). The other study reported significant alpha modulation restricted to one significant increase and one significant decrease in temporal absolute alpha power following 80 and 160 mg of ginkgo extract EGb 761, respectively (Kunkel, 1993). It is tempting to speculate that the bidirectional pattern of topographic alpha results seen in the current study may reflect a beneficial modulation of alpha activity overall.

There is some evidence of modulation by *G. biloba* of event-related potentials in sufferers from age-associated memory impairment (Semlitsch et al., 1995) and in healthy young volunteers (Luthringer et al., 1995). Whilst this was not evident in the present study, it has been suggested that the time profile of both positive and negative changes in CNV and P300 measures following ginkgo is far from straightforward (Luthringer et al., 1995). It therefore seems likely that a single 4-h postdose 'snapshot' of EEG, as in the current study, may not detect such effects.

The present study represents the first EEG investigation of a ginseng extract's ability to modulate bioelectrical cerebral activity. In the absence of any benchmark results with which to make a comparison, it seems most parsimonious to suggest that the profile of results established here following *P. ginseng* has a number of similarities to those evinced by *G. biloba*. In order to preserve the sensitivity of the statistical analysis, no direct comparison was made between the two active treatments, so it is only possible to suggest, certainly on the basis of the topographic probability maps and additional statistical evidence, that the EEG effects of this dose of *P. ginseng* (200 mg G115) are demonstrably stronger than those for the dose of *G. biloba* utilised (360 mg GK501). The effects of ginkgo were restricted to significant treatment/scalp area interactions, with significant power reductions for theta and beta bands in frontal regions. Ginseng elicited the same topographic pattern of interactions but with the addition of significantly reduced frontal alpha activity. Additionally, for ginseng, there were significant post hoc treatment effects for mean power theta and beta bands across the whole head. The relative proportion of theta activity in comparison to alpha activity across the whole scalp was also reduced more substantially by ginseng than by ginkgo (7.9% and 3.8%, respectively). It is also notable that ginseng also evinced the same pattern of alpha modulation, with frontal reductions coupled with a greater numerical increase in alpha power in the occipital region.

This raises the possibility that the effects seen here for the two extracts are dependent on the same mechanisms. It has been suggested that the beneficial effect of ginkgo on EEG is related to an up-regulation of cholinergic function (Itil et al., 1996; Luthringer et al., 1995). Cholinergic effects are documented for both ginkgo (Chopin and Briley, 1992; Huguet et al., 1994; Kristoikova and Klaschka, 1997; Taylor, 1986) and ginseng (Benishin et al., 1991; Benishin, 1992; Hsieh et al., 2000; Jin et al., 1999; Lewis et al., 1999). It is possible, therefore, that they have an effect on EEG activity through a number of cholinergic subcortical structures that are thought to underlie the rhythmicity and synchronisation of cerebral bioelectrical activity (e.g., Fisch, 1999; Steriade et al., 1993). In addition, ginkgo and ginseng share a number of other physiological effects, including antioxidant properties (Mantle et al., 2000; Siddique et al., 2000), an effect on platelet aggregation (Braquet and Hosford, 1991; Jung et al., 1998; Shi et al., 1990; Smith et al., 1996), and modulation of other haemorrheological parameters (Gillis, 1997; Jung et al., 1990; Krieglstein et al., 1986). It is possible that these or other properties allow both extracts to increase blood flow and promote increased delivery of metabolic substrates to the brain per se. However, the direct evidence of such processes is currently stronger for *G. biloba*. Given the relative strength of the effects in the two extracts, it seems certain that other neural mechanisms are having a greater influence on the EEG modulation observed here.

It is also interesting to note the fractionation of the topographic maps into 0.487 Hz intervals (1.46 Hz for beta). Inspection of these maps (Figs. 1 and 2) suggests that the modulation of activity by both extracts is restricted to specific frequencies within the conventional wavebands, and that the pattern has similarities for both extracts. As an example, in the delta band, both extracts peak modulation of activity comes in the 3.4–3.9 Hz frequency. In the theta band, both extracts exert one of their strongest decreases in power at a frequency of 7.3–7.8 Hz; and in the alpha band, both have specific decreases in the 9.3–9.8 and 13.2–13.7 Hz intervals. Whilst the relevance of these 'hotspots' of reduced activity is as yet unclear, it is possible to speculate that this decrease in specific frequency bands within summated cortical activity may reflect common regulation of the amplitude of frequency specific subcortical mechanisms.

One way in which the effect of ginseng does differ markedly from that of ginkgo in the current study is the demonstration of reduced P300 latency. Our original hypothesis, derived from the previous observation of slowed

performance on the 'speed of attention' cognitive factor following a 200-mg dose of ginseng (Kennedy et al., 2001b), was that latency would be increased. Thus, the shortened P300 latency following ginseng was somewhat unexpected and ran directly counter to our original hypothesis. Clearly, further work needs to be directed at delineating the cognitive and neural mechanisms underlying ginseng's ability to modulate cognition and bioelectric activity, and in particular the relationship between the two.

Several of the above observations and tentative interpretations are necessarily speculative. However, the current study has served to confirm that acute doses of *G. biloba* exert effects on cerebral bioelectrical activity in healthy, young volunteers. Moreover, it represents the first investigation demonstrating EEG effects following *P. ginseng*, or indeed any *Panax* species, in humans. In this respect, the demonstration of a comparatively stronger EEG effect associated with ginseng, coupled with previous demonstrations of memory improvements, suggests that the efficacy of ginseng as a cognition enhancer might usefully benefit from a similar level of adequately controlled research as that which has been directed towards *G. biloba*.

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