et al., 2004a) and between the insular and parietal cortices (Fig. 1). Since elevation of intracellular cAMP depressed oscillatory components (Fig. 2B), we presumed that depression of oscillation induction by cAMP elevation might depress intercortical strengthening between the insular and parietal cortices. When bromo-cAMP was co-applied with caffeine from the beginning, induction of oscillatory activities was depressed, but depression of oscillatory activities was incomplete. With co-application of caffeine and D-AP5 from the beginning, no oscillatory activity occurred (Fig. 1C), whereas co-application of caffeine and bromo-cAMP from the beginning yielded oscillatory activities with two or three wavelets occurring every several episodes (not shown, Fig. 2C). In addition, amplitude of the initial wavelet arriving in the parietal cortex was significantly smaller with co-application of caffeine and bromo-cAMP from the beginning (0.3 ± 0.1 mV, mean ± standard error of the mean; n = 7) than with addition of bromo-cAMP after oscillation generation in caffeine-containing medium (1.1 ± 0.1 mV; n = 7). Incomplete induction of oscillation thus generates incomplete intercortical strengthening between the insular and parietal cortices.

The next experiments investigated how application of bromo-cAMP affects establishment of insulo-parietal signal pathways by observing spatiotemporal patterns of voltage signals, using optical recording methods with voltage-sensitive dye. Electrical stimulation was delivered to the insular cortex, and optical responses were observed from the area including parietal cortex. In caffeine-containing medium, evoked signals penetrated the parietal cortex and predominantly traveled along layer II/III (Fig. 3A; upper panel). After additional application of bromo-cAMP, evoked signals predominantly traveled along layer II/III in the same way as in medium containing caffeine only (Fig. 3A; lower panel). Conversely, in medium with co-application of caffeine and bromo-cAMP from the beginning, evoked signals traveled slowly, and intensity of optical responses was weak, compared with additional application of bromo-cAMP after caffeine conditioning (Fig. 3B).

To investigate the precise spatiotemporal properties of optical signals, time courses of signal intensity recorded from region of interests (ROIs; Fig. 4A) were aligned tangentially (Figs. 4B, C). Time courses of signal intensity in the case of

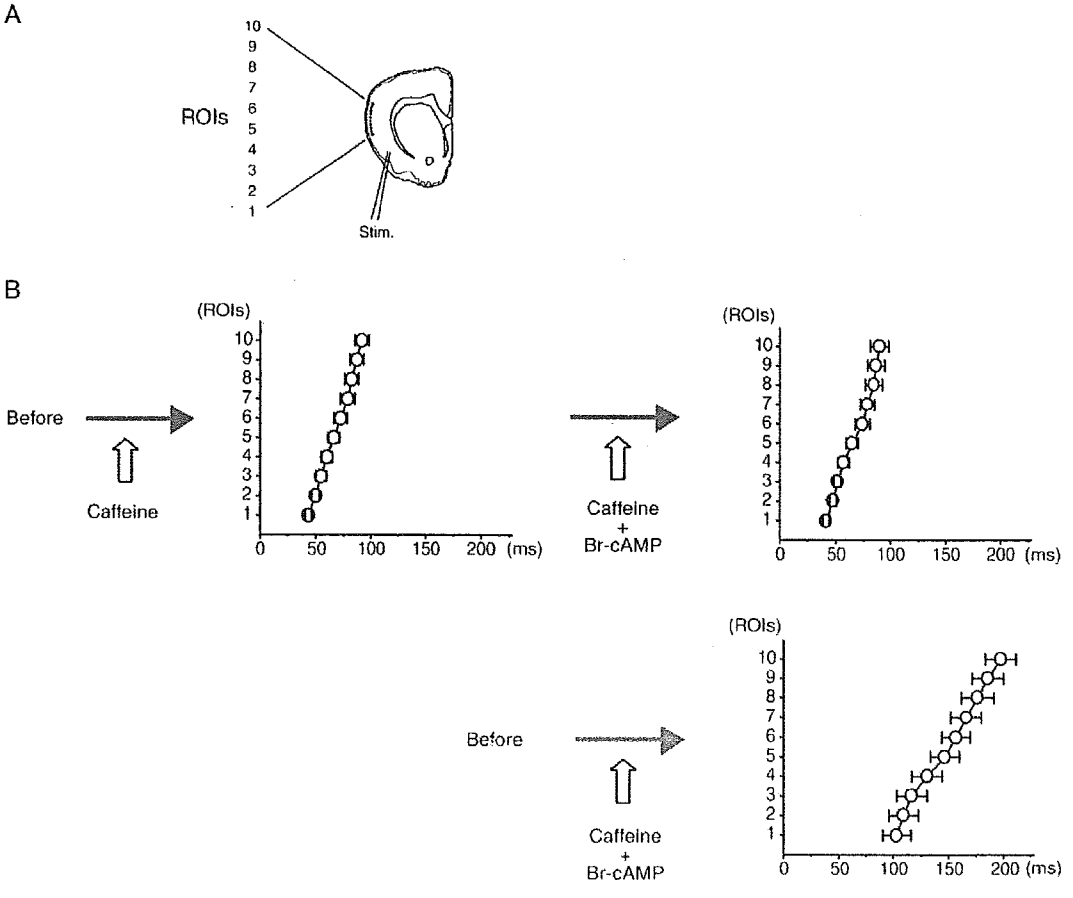

Fig. 5 – Comparing averaged time-to-peak of initial voltage signal propagation in parietal cortex under co-application of caffeine and bromo-cAMP after caffeine conditioning with time-to-peak under co-application of caffeine and bromo-cAMP from the beginning. (A) Regions of interests (ROI 1–10) are shown in an illustration of insulo-parietal cortex slice. (B) Mean time (open circle) and standard deviation (cap) were plotted. When caffeine and bromo-cAMP were co-applied from the beginning, mean time at each ROI was delayed, and mean propagating velocity was reduced (lower), compared with the case of caffeine application preceded by bromo-cAMP (upper).

caffeine application preceded by bromo-cAMP were collected from the same data displayed in Fig. 3A (upper panel). The results show that oscillation elicited by insular cortex stimulation comprises 2 components: an initial signal propagation from the insular cortex to the parietal cortex and later oscillatory signals generated in the parietal cortex that propagate bi-directionally in horizontal directions (Fig. 4B; left). This observation is consistent with the findings of our recent report (Yoshimura et al., 2004a). After addition of bromo-cAMP into the medium, initial signal propagation from the insular cortex to the parietal cortex persisted, but later waves diminished (Fig. 4B; center panel). Time courses of propagating components depressed by cAMP elevation were obtained by subtracting time courses in medium with additional bromo-cAMP from time courses in medium with caffeine alone (Fig. 4B; right panel). This clearly shows that part of the initial propagating components from the insular cortex to the parietal cortex and the greater part of oscillatory propagating components were depressed by elevations of intracellular cAMP. These results show that the dominant targets of increases in intracellular cAMP are local areas generating NMDA-receptor-dependent signal flows from the parietal cortex.

Time courses of signal intensity with co-application of caffeine and bromo-cAMP from the beginning were aligned tangentially (Fig. 4C). Time courses were collected from the same data displayed in Fig. 3B. The results show that delivery of oscillatory signals from the parietal cortex was diminished, and both amplitudes of propagating signal and propagating velocity decreased compared with caffeine application preceded by bromo-cAMP.

Time-to-peaks of the first waves recorded at ROIs over different slices were averaged. After induction of parietal oscillation in caffeine-containing medium, first waves propagated from ROI 1 to ROI 10 (Fig. 5B; upper left; $n = 7$). Mean propagating velocity of the first wave from ROI 1 to ROI 10 was 63.4 ± 8.2 mm/s. After additional application of bromo-cAMP, first waves propagated in the same way (Fig. 5B; upper right; $n = 7$), and propagating velocity was 62.7 ± 11.9 mm/s. This shows that additional application of bromo-cAMP did not

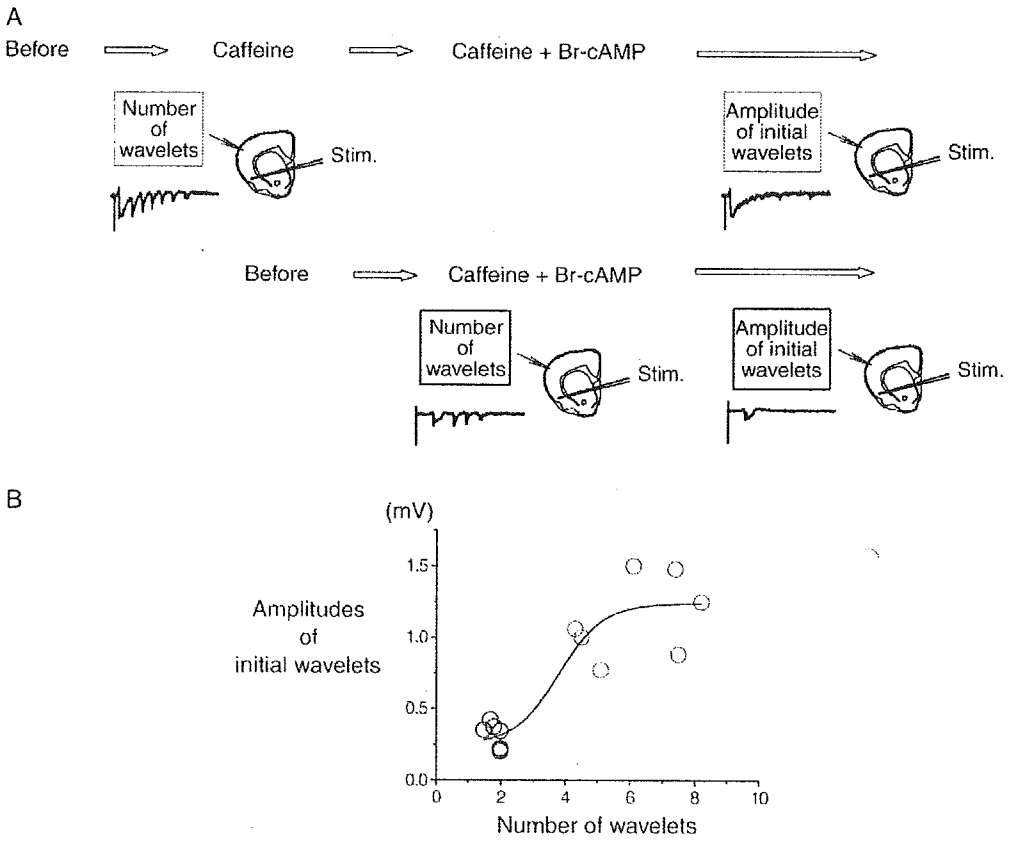

Fig. 6 – Relationship between number of wavelets of oscillatory activities after caffeine application and amplitudes of initial wavelets arriving in the parietal cortex from the insular cortex under co-application of caffeine and bromo-cAMP. (A) Schematic of timings and sites of data acquisition for preparation of part (B). Data were collected from field potential recordings, e.g., time course responses in Fig. 2. (B) Blue circle: amplitude of initial wavelets under additional application of bromo-cAMP to caffeine-containing medium was plotted against mean number of wavelets of oscillatory activities for several episodes before application of bromo-cAMP. Red circle: bromo-cAMP was bath-applied to caffeine-containing medium from the beginning. Amplitudes of initial wavelets were plotted against mean number of wavelets of oscillatory activities for several episodes. Blue squares in panel A correspond to blue circles in panel B, and red squares in panel A correspond to red circles in panel B. Note that signal propagation with sufficient amplitude requires sufficient oscillatory activities.

affect the propagating velocity of first waves. Conversely, in the case of co-application of caffeine and bromo-cAMP from the beginning, first waves managed to propagate from ROI 1 to ROI 10 (Fig. 5B; lower; $n = 7$), but propagating velocity (30.2 ± 2.7 mm/s) was significantly reduced compared with caffeine application preceded by bromo-cAMP ($P < 0.05$).

To clarify the dependence of oscillatory activities on intercortical strengthening of signal communication, relationships between number of wavelets of oscillatory activities under caffeine application and amplitudes of initial wavelets arriving in the parietal cortex from the insular cortex under co-application of caffeine and bromo-cAMP were investigated (Fig. 6). Once stable signal propagation and oscillation were established in medium with caffeine alone, initial propagation with sufficient amplitude was observed (1.1 ± 0.1 mV; $n = 7$) even after addition of bromo-cAMP. However, when caffeine and bromo-cAMP were co-applied from the beginning, induction of oscillatory activities was not stable. Oscillatory activities with 2–3 wavelets occurred every several episodes rather than every episode, and, consequently, although initial signal propagation to the parietal cortex was observed, amplitudes of initial wavelets were small (0.3 ± 0.01 mV; $n = 7$) (Fig. 2). Mean amplitude and wavelet numbers were calculated from data acquired from respective slices ($n = 7$) and plotted (Fig. 6). Incomplete induction of oscillation was seen to produce incomplete amplitude of propagating signals, suggesting a dependence of oscillatory activity on intercortical strengthening of signal communication.

3. Discussion

In the intercortical areas between primary and secondary visual cortices, the components of the horizontal pathway that is strengthened after NMDA-receptor-dependent oscillatory signal delivery using our experimental protocol are non-NMDA-receptor-dependent (Yoshimura et al., 2005b). In the same way in the present study, components of the horizontal pathway between the insular and parietal cortices that is strengthened after NMDA-receptor-dependent parietal oscillation are non-NMDA-receptor-dependent. Interestingly, increases in intracellular cAMP barely affected non-NMDA-receptor-dependent pathways that had been already strengthened but markedly diminished NMDA-receptor-dependent oscillatory activities (Figs. 2–6).

Functional glutamate receptors are expressed in both neurons and glial cells (Noda et al., 2000; Steinhäuser and Gallo, 1996). In addition, astrocytes can modulate neural synaptic activities by releasing glutamate in a Ca^{2+}-dependent manner (Parpura and Haydon, 2000). GABA receptors in inhibitory networks are modulated by activation of cAMP pathways (Cuove et al., 2002). These findings indicate that application of caffeine and bromo-cAMP might modulate synaptic activities by way of various intra- and extracellular pathways. We thus cannot even conclude that targets of cAMP under caffeine application are restricted to excitable neurons, let alone conclude that cAMP, caffeine and NMDA receptors are operating in the same neurons. However, since NMDA receptor activity-dependent neural oscillations induced by caffeine and the depression of NMDA-receptor-dependent neural activities with cAMP increases are effects produced by assemblies of various causes present in intra- and extracellular pathways, we can safely say that the dominant target of cAMP increases is NMDA-receptor-related neural activities.

The cAMP signaling pathway modulates neural functions at both channel and receptor levels, such as single channel activities (Greengard et al., 1991; Kavalali et al., 1997), probability of synaptic transmission (Abrams et al., 1991; Frey et al., 1993; Renden and Broadie, 2003; Yoshimura and Kato, 2000) and synaptic targeting of NMDA receptors (Crump et al., 2001). However, evidence that the cAMP signaling pathway regulates neural activities at the long-range intercortical network level has been lacking. In the process of establishing intercortical strengthening in our experimental model, we have described causality as follows: NMDA-receptor-dependent oscillatory signal deliveries cause strengthening of non-NMDA-receptor-dependent intercortical pathways (Yoshimura et al., 2005a,b). Thus, considering the relationship between cause and effect, increased cAMP levels directly affect the cause itself and as a result affect the effect. Since the dominant target of cAMP is considered to be NMDA receptor activities under the present experimental conditions, activation of intracellular cAMP signaling pathways can be interpreted as resulting in the induction of down-regulation in NMDA-receptor-activity-dependent strengthening of extracellular intercortical signal communications by way of diminishing NMDA-receptor-dependent oscillatory signal deliveries. This is important evidence that shows intracellular signaling pathways regulate extracellular signaling pathways between intercortical regions in the brain. NMDA receptors may act as an intermediary between actions of intracellular cAMP signaling pathways and extracellular signaling pathways at the cortical network level.

Induction of functional synapses mediated by non-NMDA receptors requires NMDA receptor activation in the hippocampus (Liao et al., 2001; Lu et al., 2001; Nicoll and Malenka, 1999). In the same way, strengthening of non-NMDA-receptor-dependent intercortical pathways in the present study required NMDA-receptor-dependent oscillatory signal deliveries from the parietal cortex. Plastic changes might thus underlie intercortical strengthening, as we previously reported (Yoshimura et al., 2005a,b). Blockade of NMDA receptor activities from the beginning completely abolished oscillatory activities and disturbed opening of signal pathways between insular and parietal cortices (Fig. 1). Compared with NMDA receptor blockade, manipulation of cAMP increases in the present experimental condition could not completely abolish oscillatory activities (Figs. 2, 6). Due to incomplete blockade of oscillation induction by cAMP increases, evoked signals from the insular cortex might manage to penetrate the parietal cortex after caffeine conditioning with increased cAMP. However, due to the attenuation of NMDA-receptor-dependent oscillatory activities by cAMP increases during caffeine conditioning, signal propagating velocity from the insular cortex to the parietal cortex in addition to amplitude of the propagating signals was reduced, compared with the case in which sufficient NMDA-receptor-dependent oscillatory signals were delivered during caffeine conditioning before manipulation of cAMP increases (Figs. 5, 6). Increases in intracellular cAMP levels could not completely abolish

opening of insulo-parietal signal communication but attenuated strengthening of insulo-parietal signal communication under caffeine application. These findings suggest that full expression of opening and strengthening of intercortical signal pathways requires sufficient NMDA-receptor-dependent oscillatory neural activities.

Caffeine displays several neuropharmacological actions (Fredholm et al., 1999; Nehlig and Boyet, 2000; Yoshimura, 2005). Among these actions, either blockade of adenosine A1 receptors or blockade of phosphodiesterases (PDEs) by caffeine induces increased intracellular cAMP levels. In general, one result of the increase in intracellular cAMP by caffeine is facilitation of synaptic activities (Fredholm et al., 1999; Moraidis and Bingmann, 1994). In the present results, however, additional increase in cAMP by bromo-cAMP application to the medium with caffeine resulted in depression of synaptic activities. A discrepancy therefore seems to exist between the actions of these manipulations, application of caffeine alone and co-application of caffeine and bromo-cAMP. However, the two manipulations produce different spatiotemporal conditions of increased cAMP and may drive a different stream of signaling pathways. As we have discussed previously (Yoshimura and Kato, 2000), determination of the direction of neuron excitability by cAMP may be dependent on the manner in which cAMP is increased.

4. Experimental procedures

All experiments were performed in accordance with the guidelines for the ethical use of animals approved by the Japanese Physiological Society. Details of the experimental procedures have been described previously by us (Yoshimura et al., 2001, 2002, 2003b, 2004a). Brains were quickly removed from Wistar rats and soaked into cold medium (2–4 °C) containing: NaCl, 124 mM; KCl, 3.3 mM; KH_2PO_4, 1.25 mM; $MgSO_4$, 1.3 mM; $CaCl_2$, 2 mM; $NaHCO_3$, 26 mM; and D-glucose, 10 mM; saturated with 95% O_2 and 5% CO_2. Coronal slices (350 μm thick) that included the insular and parietal cortices anterior to the middle cerebral artery were produced from rat brains isolated on postnatal day 27–35. Micropipettes for field potential recordings were filled with 3 M NaCl and inserted into the upper layer of the insular or parietal cortex (6–10 MΩ). Synaptic responses were evoked by single pulse stimulation at 0.03–0.3 Hz, recorded with a bridge-equipped Axoclamp-2B amplifier (Axon Instruments, Foster City, USA), digitized by an AD Digidata 1200 converter (Axon Instruments) at a rate of 2.5–5 kHz and stored in a personal computer for off-line analysis. For experimental purposes, the following drugs (purchased from Wako, Osaka, Japan) were added to medium: caffeine, 3.0 mM; D-2-amino-5-phosphonovaleric acid (D-AP5), 15 μM; 6-cyano-7-nitroquinoxaline-2,3-dione (CNQX), 20 μM; and bromo-cAMP, 40 μM.

High-speed optical recording methods with voltage-sensitive dyes were used to observe the spatiotemporal dynamics of neural activities. Details of the optical recording system used in this study have been described elsewhere (Thakur et al., 2004; Wang et al., 1999; Yoshimura et al., 2004a). Before optical recording began, slices were incubated with 0.125 mg/ml of NK2761 voltage-sensitive dye (Nihon Kanko, Okayama, Japan) for 20 min then transferred to the recording chamber. The camera unit of the optical imaging system (Fujix HR Deltaron 1700; Fuji Photo Film, Tokyo, Japan) contained a photodiode array of 128 × 128 elements. With a ×10 objective lens, the whole array corresponded to a 2.24 × 2.24 mm^2 area of tissue. Light for absorption measurements, generated by a tungsten–halogen lamp (150 W), passed through a heat absorption filter and a narrow band interference filter before being focused on the preparation. Illumination was controlled by an electromagnetic shutter to avoid dye bleaching or photodynamic damage. A run constituted the average of 16 responses elicited by cortical stimulation. Neural activity was recorded as the change in intensity of transmitted light hitting each photodiode. Images were captured every 0.6 ms. Recordings are shown as averages over 16 traces. Relative increases in optical signal were expressed as a time-sequence for the whole image or were plotted against time for each region of interest (ROI).

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