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Excitatory and inhibitory receptive fields of tectal cells are differentially modified by magnocellular and parvocellular divisions of the pigeon nucleus isthmi

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Abstract It has been known that magnocellular and parvocellular divisions of the pigeon nucleus isthmi exert excitatory and inhibitory actions on tectal cells, respectively. The present study shows that injection of *N*-methyl-D-aspartate into the parvocellular division results in an increase in responsive strength and extent of the inhibitory receptive fields, which expand into the excitatory receptive fields of tectal cells. This injection concurrently leads to a decrease in responsiveness and extent of the excitatory fields. On the other hand, injection of acetylcholine into the magnocellular division enhances visual responsiveness, although the excitatory field is not obviously changed in extent. Meanwhile, strength and extent of the inhibitory fields are decreased by acetylcholine. The excitatory and inhibitory fields are reduced in both strength and extent by magnocellular and parvocellular injection of lidocaine, respectively. It suggests that isthmic inputs from both parvocellular and magnocellular divisions converge onto the same tectal cells, and the magnocellular and parvocellular subnuclei can modulate excitatory and inhibitory receptive fields of tectal cells, respectively, with some interactions between both fields.

Key words Receptive field · Optic tectum · Nucleus isthmi · Pigeon · Visual system

Abbreviations *GABA* gamma-aminobutyric acid · *ERF* excitatory receptive field · *Imc* nucleus isthmi pars magnocellularis · *Ipc* nucleus isthmi pars parvocellularis · *IRF* inhibitory receptive field · *IS* index of suppression · *NMDA N*-methyl-D-aspartate

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Introduction

The receptive field of a visual cell is an area on the retina or in space, stimulation of which modifies the excitability of the cell. In birds, the receptive field of tectal cells is usually composed of an excitatory center and an inhibitory surround (Frost et al. 1981; Hughes and Pearlman 1974; Jassik-Gerschenfeld and Guichard 1972; Jassik-Gerschenfeld et al. 1975; Jassik-Gerschenfeld and Hardy 1979). The excitatory receptive field responds to moving spots (Frost et al. 1981; Jassik-Gerschenfeld and Guichard 1972; Jassik-Gerschenfeld et al. 1975), gratings (Frost et al. 1981; Jassik-Gerschenfeld and Hardy 1979), kinematograms (Frost et al. 1988) and color patterns (Sun and Frost 1997). It has been suggested that electrophysiological properties of tectal cells are modulated by input from extratectal sources such as the visual wulst and the nucleus isthmi (Bagnoli et al. 1979).

The nucleus isthmi in birds is a visual center (Wang and Frost 1991; Yan and Wang 1986), which is divided into two divisions, the nucleus isthmi pars parvocellularis (Ipc) and the nucleus isthmi pars magnocellularis (Imc). They receive input from the ipsilateral tectum (Hunt et al. 1977; Reiner and Karten 1982; Woodson et al. 1991) and project back to the tectum (Hunt et al. 1977; Tömböl and Németh 1998; Wang and Wang 1990). Within the tectum, the axons of Ipc and Imc cells give off broad branches in a columnar fashion (Hunt et al. 1977; Tömböl 1998; Tömböl and Németh 1998). Electron microscopic observations have indicated that isthmotectal terminals form synapses with tectal dendrites in amphibians (Gruberg et al. 1994). This is the case with the parabigeminal nucleus, which is the mammalian homologue to the nucleus isthmi. Its parabigeminotectal terminals are presynaptic to dendrites within the superior colliculus (Feig et al. 1992).

Though several immunohistochemical studies have demonstrated the presence of gamma-aminobutyric acid (GABA)-immunoreactivity in all Imc cells of the chicken (Tömböl and Németh 1998) and the pigeon (Veenman

and Reiner 1994), but in none of the Ipc cells in both species (Granda and Crossland 1989; Tömböl and Németh 1998; Veenman and Reiner 1994), electrophysiological studies have shown that visual responses of tectal cells in pigeons are modulated by Ipc in an inhibitory manner and by Imc in an excitatory way (Wang et al. 1995a). The Ipc efferents to tectum are mainly GABAergic, acting via $GABA_A$ receptors on postsynaptic tectal cells (Felix et al. 1994; Gao et al. 1995a), whereas the Imc efferents to tectum are either cholinergic, via muscarinic receptors, or glutamatergic, mediated mainly by NMDA (*N*-methyl-D-aspartate) receptors (Wang et al. 1995b). Moreover, acetylcholine affects almost equally neuronal firing in Ipc and Imc, but NMDA-induced discharge is specifically restricted to Ipc (Wu et al. 1994). This difference provides a good model in which to use NMDA to excite Ipc cells without any action on Imc cells.

The receptive field of isthmic cells in pigeons may be formed by feedforward convergence of tecto-isthmic input from a group of tectal cells with receptive fields consisting of excitatory centers and inhibitory surrounds (Wang and Frost 1991), although data are needed to show how the receptive field of tectal neurons is formed or modified by feedback pathways from the isthmic complex to tectum. Therefore, the present study was undertaken to elucidate the effects of chemical excitation and blockade of Ipc and Imc on the extent and responsiveness of the excitatory center and inhibitory surround of the receptive fields of tectal cells in pigeons.

Materials and methods

The experiments were performed on 27 adult pigeons (*Columba livia*) of either sex, weighing 265–410 g, under guidelines regarding the care and use of animals, approved by the Society for Neuroscience. The pigeons were anesthetized with urethane (20%, 1 ml/100 g body weight, i.m.), and then placed in a stereotaxic apparatus. Body temperature was maintained at 41 °C by a heating pad. The left tectum was surgically exposed and the overlying dura mater excised. The nictitating membrane of the right eye was removed and the eye kept open. The left eye was occluded with an opaque cover. A screen was positioned 40 cm distant from the viewing eye, and 24° to the midsagittal plane of the pigeon; it measured 180 cm in height and 220 cm in width. The area being visually stimulated was about 140° (horizontal angle) by 130° (vertical angle). In view of the fact that the angle between the horizontal meridian of the visual field and the eye center-bill tip line of the stereotaxically fixed pigeon was 72° in these experiments, whereas it is 34° for normal flying, walking, standing and perching (Erichsen et al. 1989), the horizontal meridian was rotated clockwise by 38° (Fu et al. 1998).

A two-barrel micropipette (Li et al. 1998) was used both for recording Ipc or Imc cells following the pigeon's brain atlas (Karten and Hodos 1967), and for marking the recording sites. One of the barrels was filled with 2 mol l^{-1} NaCl and 100 mmol l^{-1} $CoCl_2$ for recording and marking, and the other connected to a pneumatic micropump (World Precision Instruments, Sarasota, Fla., PV800) contained acetylcholine chloride (ACh, 0.2 mol l^{-1}, pH 6.7, Sigma Chemical, St. Louis, Mo.) or NMDA, 0.05 mol l^{-1}, pH7.5, Sigma Chemical), or lidocaine hydrochloride (2%, Dongting Pharm., Hunan, China) for exciting or blocking the isthmic cells, whose excitatory receptive fields (ERFs) overlapped (30–40°) those of tectal cells to be examined. In 3 of 27 pigeons, the two-barrel pipette was used to sequentially find both Ipc and Imc cells in the same penetration whose ERFs overlapped those of tectal cells, in order to examine whether isthmic afferents from both Ipc and Imc converge onto the same tectal cells. Action potentials of tectal cells were extracellularly recorded with an additional micropipette containing 2 mol l^{-1} NaCl and 100 mmol l^{-1} $CoCl_2$, which was advanced approximately perpendicular to the brain surface (Fig. 1). All visual responses were fed into the computer for on-line analysis.

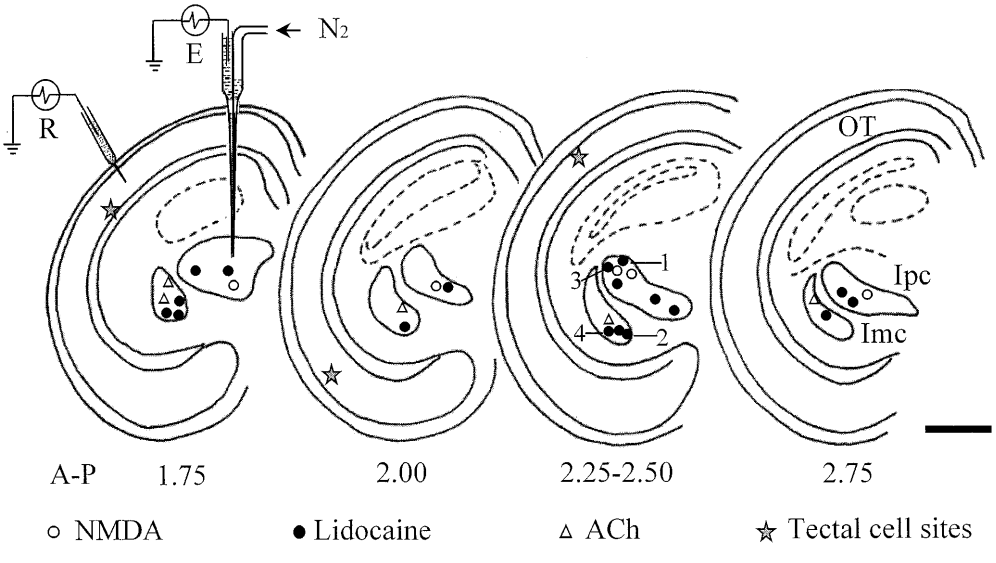

Fig. 1 Serial sections of the pigeon midbrain at levels of the nucleus isthmi pars parvocellularis (*Ipc*) and the nucleus isthmi pars magnocellularis (*Imc*) showing experimental arrangement of a two-barrel micropipette (*E*) in the isthmic complex, a recording electrode (*R*) in the optic tectum (*OT*), and the distribution of drug injection sites marked with cobalt sulfide. *Solid circles* represent lidocaine injection sites; *empty circles and triangles* symbolize injection sites of *N*-methyl-D-aspartate (NMDA) within Ipc and of acetylcholine within Imc, respectively. Numbers 1–2 and 3–4 show injection sites sequentially marked within Ipc and Imc in the same penetrations. *Stars* represent marked sites of tectal cells. Numerals beneath sections are anterior-posterior (A-P) levels according to the pigeon's brain atlas (Karten and Hodos 1967). N_2 nitrogen gas for pneumatic injections. Bar: 2 mm

Two types of visual stimuli were generated by a workstation (Indigo 2, Silicon Graphics, Mt. View, Calif.) and rear-projected by a three-color projector (Electrohome ECP4101, Electrohome, Kitchener, Ontario, Canada) onto the screen. Type 1 was a black square, and type 2 was black twin-squares. These squares measured 2.3–4.0° in dimension, with luminance of 2.0 cd m^{-2}, and moved at 20.7–36.4° s^{-1} against a white background of 14.2 cd m^{-2}. The illumination of the room was about 35 lx (Gao et al. 1995b). The single square was moved along numerous parallel trajectories (2–4° spacing) covering the whole screen (Fu et al. 1998) in four to eight directions (0° nasal, 45°, 90° dorsal, 135°, 180° temporal, 225°, 270° ventral, 315°), and visual responses were fed into the computer for plotting an ERF. For silent cells, the extent of the ERF was taken as the region from which discharges were evoked; for spontaneously active cells, it was the region from which responses larger than the average spontaneous rate were evoked. The twin-squares technique was used as Frost et al. (1981) described for plotting the extent of an inhibitory receptive field (IRF): while the first square was moved along the ERF center-line, the second one was moved in the surrounding region with increasing distance (2–4° spacing) from the ERF outer perimeter in four to eight directions. The IRF outer perimeter was determined by an index of suppression (IS) of 75 that was obtained from statistic analysis of six tectal cells. The IS was defined here as the number of spikes produced by the twin-squares divided by that of spikes evoked by the single square, multiplied by 100. Therefore, the IS values ranged from 0 (inhibition was strongest) to 100 (inhibition was weakest).These stimuli were also used for examining effects of chemical excitation or blockade of Ipc and Imc on the extent or responsiveness of receptive fields of tectal cells. In doing these examinations, the first square was moved along the ERF center-trajectory and the second one moved along a trajectory parallel to the first stimulus trajectory and 4–15° distant from the ERF outer perimeter. The spacing between trajectories of the two stimuli was so chosen that the second stimulus produced the largest surround effect in the control condition. This spacing was fixed for a given cell, but varied from cell to cell.

To mark the drug injection sites within Ipc and Imc, and occasionally the recording sites in tectum, cobalt ions were ejected with rectangular current pulses of 10–20 μA in intensity and 0.5 s in duration for 10–15 min. The brain was removed from the skull and immersed for 10–15 min in a 10% ammonium sulfide solution to form a black precipitate (CoS). The brain block was then fixed for 6–12 h in 4% paraformaldehyde, soaked in 30% sucrose solution overnight. Frozen sections were cut at 100 μm thickness, and counterstained with cresyl violet. Sections were dehydrated and covered for microscope observation (Li et al. 1998; Wang et al. 1981).

Results

Thirty-three tectal cells were examined for effects of chemical excitation or blockade of Ipc or Imc on the extent and responsiveness of their receptive fields, each of which was composed of an ERF surrounded by an IRF. These receptive fields were generally oval-shaped and mainly localized in the anterior-inferior visual field. The extent of the central ERF ranged from 8° to 94° (mean = 42°) and that of the peripheral IRF ranged from 64° to 114° (mean = 81°). According to the micromanipulator depth reading, corrected by three cobalt markings within tectum (±80 μm), the recording depth of tectal cells ranged from 480 μm to 1380 μm, which mainly corresponded to the stratum griseum et fibrosum superficiale and the stratum griseum centrale. Drug injection sites marked with cobalt in 24 pigeons were all localized within Ipc (13 sites) and Imc (11 sites). Cobalt markings were also made in two of three additional pigeons with overlapping ERFs of visual cells in tectum, Ipc and Imc, showing that drug injection sites in the same penetrations were localized within Ipc and Imc (Fig. 1).

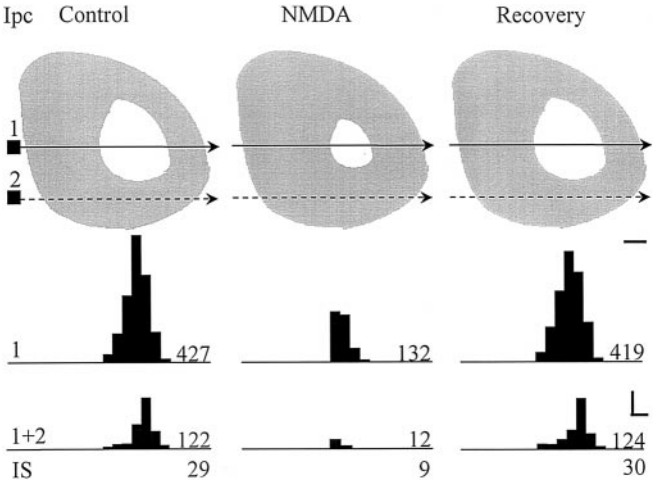

Fig. 2 Effects of NMDA (10 nl) injected into Ipc on the receptive field profiles and visual responses of a cell 988 μm deep in the tectum. During NMDA injections, visual responsiveness was reduced, and the excitatory receptive field (ERF) was gradually diminished and taken over by the expanding inhibitory receptive field (IRF). Stimulus 1 was moved at velocity of 19.4° s^{-1} through the ERF in the dorsoventral direction (*solid arrow*) to examine visual responses, and stimulus 2 was moved through the IRF (*dotted arrow*) with stimulus 1 in the ERF (1 + 2) to examine inhibitory responses. Histograms of visual responses are shown beneath the corresponding field profiles. *Numerals* to the right of histograms are total numbers of spikes accumulated for three sweeps, and those beneath the histograms are IS (index of surround suppression) values, indicating an increase in inhibitory strength. *White and gray areas* represent ERF and IRF, respectively. Bar: 10°; scales in histograms: 400 ms, 30 spikes

Effects of injecting NMDA (10–60 nl) into the Ipc on the extent and responsiveness of receptive fields were examined on seven tectal cells recorded from five pigeons. Drug action usually started 30 s after its application and reached maximum in 1–4 min. Tectal IRFs expanded inward the ERFs and took over about half of the ERF extent, without observable change in inhibitory circumference, and their inhibitory strength increased, showing a change of IS values from 43.5 ± 18.2 to 6.9 ± 5.8 (mean ± SD, $n = 7$). Meanwhile, visual responses of these cells were reduced to 30.9 ± 16.0% ($n = 7$) of control level, and ERF shrunk to 54 ± 23.8% ($n = 4$) of their pre-drug extent (Fig. 2). These effects recovered 3–10 min after stopping drug application. On the other hand, injection of lidocaine (20–80 nl) into Ipc in eight pigeons resulted in shrinkage of the IRF toward the ERF and the IRF finally disappeared in four of eight cells examined, leaving the "exposed" ERF alone (Fig. 3). The IS values of these IRFs changed from 21.5 ± 17.1 in controls to 90.9 ± 15.7 during lidocaine inactivation ($n = 8$). Concurrently, ERF responsiveness reduced to 89.7 ± 10.6% ($n = 6$) of control level, although the ERF was not usually reduced in size. Drug

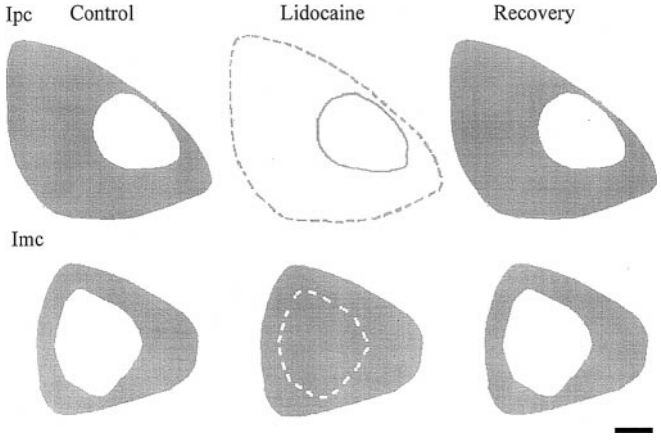

Fig. 3 Lidocaine (50 nl) injected into Ipc (*upper*) or Imc (*lower*) exerted differential actions on the receptive field profiles of two tectal cells with recording depths of 726 μm and 1138 μm, respectively. In the first case, lidocaine within Ipc resulted in reduction of inhibitory strength and final elimination of IRF (*dotted circle*) with the ERF exposed (*solid line circle*). In the second case, lidocaine within Imc reduced visual responsiveness and expanded the IRF to the ERF. *White and gray areas* represent ERF and IRF, respectively. Bar: 20°

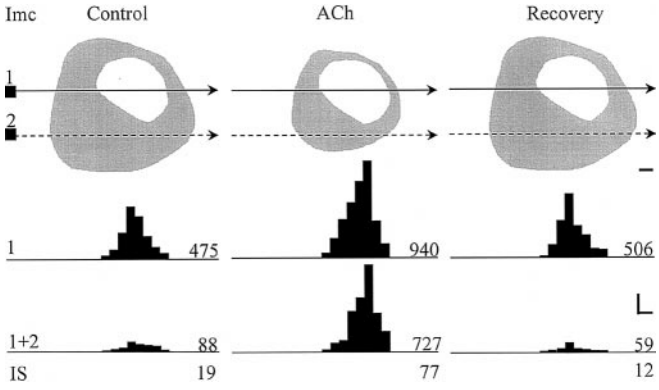

Fig. 4 Effects of acetylcholine (20 nl) injected into Imc on the receptive field profiles and visual responses of a cell 1220 μm deep in the tectum. During acetylcholine injection, visual responsiveness of the ERF was enhanced but its extent was not obviously changed, whereas the extent and inhibitory strength of the IRF were reduced. Stimulus 1 was moved at velocity of 22.8° s^{-1} (*solid arrow*) through the ERF in the temporonasal direction to examine visual responses, and stimulus 2 was moved (*dotted arrow*) through the IRF together with moving stimulus 1 in the ERF (1 + 2) to examine inhibitory responses. Histograms are shown beneath the corresponding field profiles. *Numerals* to the right of histograms are total numbers of spikes accumulated for three sweeps and those beneath the histograms are IS values indicating a decrease in inhibitory strength. *White and gray areas* represent ERF and IRF, respectively. Bar: 10°; scales in histograms: 400 ms, 60 spikes

action started 30 s post-application and reached maximum in 60–180 s. Effects of lidocaine on receptive fields were usually eliminated 4–12 min after stopping drug application.

Effects of Imc excitation on receptive fields were examined on seven tectal cells recorded from five pigeons. Due to the presence of acetylcholine receptors and the lack of NMDA receptors within Imc (Wu et al. 1994), acetylcholine (10–80 nl) was injected into the Imc to excite the Imc efferents to tectum. Drug action started 30 s after application and reached maximum in about 90 s. Following acetylcholine application, visual responses of these cells increased to 266 ± 114% ($n = 7$) of pre-drug level, and ERFs did not show observable changes in size. Simultaneously, acetylcholine reduced the strength of the IRFs of these cells, as characterized by a change of IS values from 22.4 ± 12.8 in control trials to 61.4 ± 10.5 ($n = 3$) during acetylcholine application. These cells' IRFs gradually shrunk toward their ERFs, with the IRF in one of these cells being completely abolished (Fig. 4). The extent and responsiveness of tectal cells returned to control levels 3–10 min after stopping drug application. On the other hand, lidocaine (20–80 nl) injected to block Imc-tectal transmission had just opposite effects on tectal cells. In 30–120 s after lidocaine application, visual responses of eight cells recorded from six pigeons were reduced to 23.7 ± 13.1% ($n = 8$) of control level. Effects of lidocaine injected into the Imc on ERF extent were examined in two tectal cells. The ERF *area size* in one cell was reduced to about 70%, and that in the other reduced to about 20% of pre-drug size (Fig. 3). The IRF expanded inward to take over about 30–80% of the ERF, and its inhibitory strength was not obviously changed in two of the three cells examined, with the other cell's IS value changing from 40.8 to 96.6. The IRF peripheral dimension did not show any observable changes. These effects went back to normal 3–10 min after ceasing drug application.

To further provide evidence that both Ipc and Imc cells converge onto the same tectal cells, we used a two-barrel pipette to sequentially isolate an Ipc cell first and then an Imc cell in the same penetration, with overlapping ERFs. An additional pipette was then used to isolate a tectal cell whose ERF overlapped the ERFs of both the Ipc and Imc cells by 30–40°. For three tectal cells with triple receptive field overlap, their visual responsiveness was reduced to an average 15% of control level, and the size of the ERF reduced to an average 42% of its pre-drug size by lidocaine (50 nl) injected into the Imc. Following injection of lidocaine (50 nl) into Ipc, IS values were changed from 14 to 78, and the IRF was reduced to an average 24% of its pre-drug *area size*. These effects were correspondingly similar to those obtained by examining the receptive field properties of tectal cells whose receptive fields overlapped those of Imc or Ipc cells during lidocaine injection into either Imc or Ipc.

Discussion

One major finding of the present study is that the excitatory center and inhibitory surround of the receptive fields of tectal cells in pigeons are differentially modified by the magnocellular and parvocellular divisions of the nucleus isthmi, respectively. This finding is supported by the previous studies showing that Imc-efferents to tectum are excitatory (Wang et al. 1995a, b), whereas

Ipc-efferents to tectum are primarily inhibitory (Felix et al. 1994; Gao et al. 1995a; Wang et al. 1995a). However, it is apparently in disagreement with several immunohistochemical studies which show that GABA-immunoreactivity is stained in all Imc cells of the chicken (Tömböl and Németh 1998) and the pigeon (Veenman and Reiner 1994), and in none of Ipc cells in both species (Granda and Crossland 1989; Tömböl and Németh 1998; Veenman and Reiner 1994), and that Ipc cells in pigeons are immunostained for choline acetyltransferase (Bagnoli et al. 1992; Sorenson et al. 1989) and acetylcholinesterase (Li et al. 1987) suggestive of cholinergic nature of the Ipc-tectal projection. Nevertheless, these are not fully supported by other studies which demonstrate GABA-immunoreactivity in about 40% of Imc cells and in neurons within the rostro-medial Ipc in pigeons (Wang et al. 1993). An autoradiographic study (Hunt et al. 1977) has shown that a small group of cells in the rostral Ipc are GABAergic and those in the remaining part of the pigeon Ipc might be glycinergic. These discrepancies may be due to differences in species and methodologies used by various authors. The inconsistency of the present results with those obtained by the above-mentioned studies could be explained by the possibilities: GABA-positive Imc cells (40%) might be inhibitory interneurons in the pigeon (Wang et al. 1993) but not in the chicken (Tömböl and Németh 1998), and other GABA-immunonegative neurons are tectally projecting cells, which are either glutamatergic or cholinergic (Wang et al. 1995b). Tectally projecting Ipc neurons are cholinergic (Bagnoli et al. 1992; Li et al. 1987; Sorenson et al. 1989), about 20% of which can evoke cholinergic excitation followed by GABAergic inhibition in tectal cells and 80% others inhibit tectal cells (Felix et al. 1994; Gao et al. 1995a) probably by cholinergic activation of intrinsic GABAergic systems within tectum (Felix et al. 1994; Hunt and Künzle 1976). Furthermore, GABA-positive cells in the rostral Ipc (Hunt et al. 1977; Wang et al. 1993) could directly inhibit tectal neurons. It is also noteworthy that biochemical, neuroanatomical and physiological criteria should be met altogether to finally identify a substance as a neurotransmitter. For example, high concentration of acetylcholinesterase is a necessary but not sufficient condition for identifying cholinergic neurons (Lehmann and Fibiger 1979).

In view of a differential distribution of NMDA and acetylcholine receptors within Ipc and Imc (Wu et al. 1994), we therefore used NMDA and acetylcholine to excite Ipc and Imc cells, respectively. Excitation of Ipc cells by NMDA results in enhancement of inhibitory strength and inward expansion of IRFs due to activation of the Ipc-tectal pathway, which is inhibitory and mediated mainly by GABAergic synapses on tectal cells (Felix et al. 1994; Gao et al. 1995a). A decrease in visual responses of tectal cells in this situation may stem from three sources: (1) inhibition by activation of the Ipc-efferents to tectum (Felix et al. 1994; Gao et al. 1995a); (2) reduction in the extent of the ERF by an inward expanding IRF; and/or (3) increase in long-range inhibition due to a longer distance the stimuli have to pass through the expanded IRF (Uchiyama et al. 1998). On the other hand, injection of acetylcholine into Imc leads to enhancement of ERF excitability due to excitation of the Imc-tectal pathway, which is excitatory and mediated by glutamatergic and cholinergic synapses on tectal cells (Wang et al. 1995a, b). A decrease in inhibitory strength of tectal cells under this condition could be explained by two possibilities: (1) enhancement of ERF responsiveness by acetylcholine results in an apparent decrease in inhibitory strength (IS value) of IRF; or (2) long-range inhibition (Uchiyama et al. 1998) is decreased due to reduction of IRF size. This IRF shrinkage probably results from the fact that Imc-axon collaterals to Ipc-neurons (Tömböl et al. 1995; Tömböl and Németh 1998) play an inhibitory role within the Ipc, or, alternatively, the IRF size is reduced by excitability-enhanced ERF through competition.

It has been known that lidocaine is a local anesthetic which operates as a sodium channel blocker. Lidocaine injected into Imc and Ipc can block afferents from Imc and Ipc to tectum, and thereby diminish or abolish ERF and IRF, respectively. These blockade effects convincingly indicate that isthmic inputs are essential for modulation of receptive fields of tectal cells and for retinotectal transmission in birds. It is interesting to note that the present study satisfactorily explains the puzzle of why lidocaine injected into Ipc did not work (Wang et al. 1995a), because blockade of the Ipc efferents to tectum by lidocaine eliminates the inhibitory surround but not the excitatory center of receptive fields of tectal cells, and no obvious effects were therefore observed on visual responses of tectal cells.

It is worth mentioning that effects exerted by NMDA, acetylcholine and lidocaine are, for three reasons, pharmacological but not toxicological. First, because of the small amounts of drugs and the great distance from the nucleus to the tectal cells examined, drugs can not spread directly to tectal cells (Wang et al. 1995a). Second, drugs show their effects on the extent and responsiveness of receptive fields of tectal cells in a topographical manner, characterized by the fact that receptive fields of tectal cells have to overlap those of Ipc and/or Imc cells in order to show pharmacological effects (Wang et al. 1995a). Third, the specific physiological effects of these chemicals on the excitatory center and inhibitory surround of receptive fields of tectal cells depend both on the kind of drugs used and the division of the nucleus injected.

The second major finding of the present study is that both Imc and Ipc cells converge onto the same tectal cells. This statement is based on the two facts: first, the receptive field of *every* tectal cells whose receptive fields overlap those of either Imc or Ipc cells at the drug injection site was modified by chemical excitation or blockade of either Imc or Ipc. This is in accordance with the previous finding that visual responses of *every* tectal cells are depressed by injecting lidocaine into Imc or NMDA into Ipc (Wang et al. 1995a), and affected by

electrical stimulation of either Imc or Ipc (Felix et al. 1994; Gao et al. 1995a; Wang et al. 1995b). This implies that simultaneous manipulation of both Imc and Ipc is likely to demonstrate effects of both divisions on the same tectal cells. Second, receptive fields of the same tectal cells can be modified in size and/or responsiveness by chemical injections sequentially into Imc and Ipc, on condition that receptive fields of tectal cells overlap those of both Imc and Ipc cells. Due to the limited space, it is difficult to use two electrodes to inject drugs into Imc and Ipc separately, so we used the same pipette to inject drugs into Ipc and then Imc in the same penetration. However, the successful rate of finding Ipc and Imc cells in the same penetrations whose receptive fields overlap is only about 10% in the present study. Therefore, we can not examine many tectal cells in this way due to technical difficulty. The convergence of isthmic afferents onto tectal cells is also supported by several anatomical studies showing that within the avian tectum, Ipc and Imc axons give off broad branches in a columnar fashion (Hunt et al. 1977; Tömböl 1998). The isthmotectal terminals may make axo-axonal synapses with retinotectal terminals, which in turn contact tectal cells, or be interposed between retinotectal fibers and dendrites of tectal cells, or share the same target with retinotectal axons (Wiggers 1998). Electron microscopic observations have indicated that isthmotectal terminals make synapses with tectal dendrites in amphibians (Gruberg et al. 1994). This is the case with the parabigeminal nucleus, the mammalian homologue to the nucleus isthmi. Its parabigeminotectal terminals are presynaptic to dendrites within the superior colliculus (Feig et al. 1992).

It has been suggested that receptive fields of isthmic cells in birds may be formed by convergence of those of tectal cells (Wang and Frost 1991). The present study provides strong evidence indicating that the excitatory center and inhibitory surround of receptive fields of tectal neurons in pigeons are differentially modified by Imc and Ipc, respectively. It is conceivable that excitatory and inhibitory circuits intrinsic to tectum may also play important roles in modulating receptive fields of tectal neurons. Our findings lend support to the notion that receptive fields of visual neurons are a dynamic structure, sub-regions of which strongly interact or compete with each other.

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