Pharmacological Reports
2007, 59, 408–413
ISSN 1734-1140

Regulation of serotonin N-acetyltransferase activity in the chick pineal gland by UV-A and white light: role of MK 801- and SCH 23390-sensitive retinal signals

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Abstract:
The rhythmic melatonin synthesis in the pineal gland is one of the most extensively studied circadian rhythms in vertebrates. Light is the dominant environmental factor controlling this process. Light at night acutely suppresses pineal melatonin content and activity of serotonin N-acetyltransferase (AANAT; the key and penultimate enzyme in the hormone biosynthetic pathway). In addition, pulses of light appropriately timed reset the circadian oscillator generating the melatonin rhythm. Although the avian pineal gland is a directly photosensitive organ, it has recently been demonstrated that light perceived by the eyes only regulates its activity. The present study shows that ocular exposure of chicks to UV-A radiation or white light during the second half of the subjective night markedly decreased AANAT activity in the pineal gland, and produced a significant phase advance of the circadian rhythm of the enzyme activity. Both the suppressive and phase-shifting effects of UV-A light were antagonized by intraocular pretreatment of birds with MK 801 (a selective blocker of NMDA glutamate receptors), but were not modified by SCH 23390 (a selective antagonist of D1-dopamine receptors). On the other hand, the suppressive and phase-shifting effects of retinally perceived white light were antagonized by intraocular injection of SCH 23390, and not affected by MK 801. Our results demonstrate that retinal illumination with UV-A radiation and white light provide powerful signals that shift phase of the circadian oscillator generating melatonin rhythm in the chick pineal gland. It is suggested that control of pineal melatonin synthesis by retinally perceived UV-A and white light might involve input from different photoreceptors.

Key words:
melatonin, AANAT, pineal gland, circadian rhythm, light, dopamine, glutamate, chick

Abbreviations: AANAT – serotonin N-acetyltransferase, CT – circadian time, DD – constant darkness, LD – light-dark, UV – near-ultraviolet

Introduction

Circadian rhythms constitute a common and fundamental feature of living systems. They are generated by an endogenous oscillator(s) and timed through the work of a pacemaker [24]. The characteristic feature of a circadian rhythm is its persistence in constant conditions (namely the constant darkness, DD) and entrainment by cyclic environmental stimuli, with light being the most powerful entraining stimulus [24]. One of the most extensively studied circadian rhythms in vertebrates is that of melatonin, an indoleamine hormone produced by the pineal gland, and

additionally in some species, by the retina [4, 17, 33]. The biosynthesis of melatonin occurs in a light-dependent rhythmic fashion controlled by an endogenous circadian clock, with high levels at night, and low levels during the daytime [4, 17, 30, 33]. The rate of melatonin formation is regulated primarily by the activity of serotonin N-acetyltransferase (arylalkylamine N-acetyltransferase; AANAT), the penultimate enzyme in the hormone biosynthetic pathway [14].

Light is the dominant environmental factor controlling melatonin biosynthesis, and as such it exerts two distinct effects on the hormone production. Light at night acutely suppresses AANAT activity and melatonin content. In addition, pulses of light appropriately timed reset the circadian oscillator generating the melatonin rhythm in a phase-dependent manner [4, 17, 28, 30, 33]. In mammals, photic regulation of pineal activity involves stimulation of retinal photoreceptors with subsequent activation of a multisynaptic pathway conveying the light information downstream from the retina to the pineal gland [4, 33]. In contrast to mammals, the avian pineal gland is a directly photosensitive organ (e.g. [20, 21, 26, 28]), and light acting *via* the skull has been shown to suppress pineal melatonin production [1, 5, 35]. In addition, it has recently been demonstrated that light perceived by the retina only acutely inhibits melatonin production in the chicken pineal gland [9, 18, 19, 25, 35] and entrains the circadian rhythm of AANAT activity and melatonin in the pineal gland of chicken and Japanese quail [5, 9, 34]. The suppressive effect has been shown to result from retinal illumination with visible, full spectrum white light as well as with near-ultraviolet radiation (UV-A; λ_{max} = 365 nm), and involved stimulation of retinal D1-dopamine and NMDA glutamate receptors, respectively [9, 18, 25, 35]. The present study was undertaken to investigate whether UV-A light acting on the eyes only is also capable of phase shifting the biological oscillator generating the circadian rhythmicity in the chick pineal gland. Thus, the effect of ocular exposure of chicks to UV-A light on the circadian rhythm of AANAT activity in the pineal gland was analyzed, and compared with the action exerted by white light. In addition, an involvement of retinal NMDA glutamate and D1-dopamine receptors in the studied processes was examined.

Materials and Methods

Animals

Experiments were performed on 8–14 days old male white leghorn chicks (*Gallus domesticus*; Hy-Line) purchased locally on the day of hatching, and kept in temperature-controlled warmed brooders with *ad libitum* standard food and tap water. The animals were entrained to a 12 h light : 12 h dark illumination (LD) cycle for a minimum of one week prior to the study. The lighting cycle was produced by overhead cool fluorescent lamps providing light intensity at the level of the animals' heads of approximately 150 lux. The experiments were carried out in strict accordance with the Polish governmental regulation concerning experiments on animals (Dz.U.05.33.289). All the experimental protocols were approved by the Local Ethical Committee for Experimentation on Animals.

Chicks, adapted to the LD cycle, were transferred into constant darkness (DD) at the time of normal light-to-dark transition. On the second day of DD chicks were divided into four experimental groups: (A) control – kept in DD throughout the study; (B) light-exposed – animals exposed to light for one hour, between CT21 and CT22, and then returned into DD; (C) drug-treated – at CT20 chicks received intraocular (*ioc*) injection of MK 801 or SCH 23390 into both eyes; (D) drug-treated and light-exposed – one hour prior to the light exposure the animals were injected *ioc* with MK 801 or SCH 23390. CT20 refers to the beginning of the ninth hour of the subjective dark phase of the DD cycle. The *ioc* administration was accomplished by a slow (10 s) injection of 10 µl of appropriate solution into the vitreous body using a 30-gauge needle and 25-µl Hamilton syringe, under a short-acting (up to 2 min) ether anesthesia. In Experiment 1, the animals were exposed to near-ultraviolet light (UV-A). UV-A light (λ_{max} = 365 nm, 28 nm half-peak bandwidth) was produced by 16 W VL-204 BLB lamp (2 × T-4LN tube; Vilber Lourmat, Marne la Vallee, France) and passed through a UFS-6 filter. The light source was fixed 50 cm above the animals' heads to provide irradiance of 10 µW/cm^2 at the eyes level (measured by UVR 365 Radiometer, Vilber Lourmat, Marne la Vallee, France). In Experiment 2 chicks were exposed to white light of 4 lux intensity at the level of the animals' heads. To ensure only retinal illumination, during the light exposure the heads of chicks were covered with black opaque tape (done

on the first day of DD). The head patches (30 mm in diameter) were centred over the pineal region of the brain. Chicks from all experimental groups were decapitated at regular time intervals over a 24 h period, the decapitation begun in the middle of the subjective light phase (CT6) on the third day of DD. To evaluate the acute suppressive action of light pulse on pineal AANAT activity and effects of the drug treatment upon it, additional groups of animals were killed immediately after the light exposure and 2 h after the *ioc* injection of the drugs. Pineal glands were quickly isolated, frozen on dry ice, and stored at –70°C until assayed. The drugs were dissolved in 0.9% NaCl immediately before use. All injections and tissue dissections were performed under dim red light (3 lux).

Pineal glands were sonicated in ice-cold 0.05 M sodium phosphate buffer (pH 6.8) in the proportion of 1 pineal/100 μl. The homogenate was centrifuged at 10,000 g for 5 min at 4°C and aliquots of the supernatant were assayed for AANAT activity. AANAT activity was determined according to the radioisotopic method previously described [23], using as substrates acetyl coenzyme A (152 μM) containing 16 nCi of [acetyl-1-^{14}C]coenzyme A and tryptamine–HCl (1.5 mM).

Chemicals

[Acetyl-1-^{14}C]coenzyme A (sp. act. 60 mCi/mmol) was purchased from Perkin-Elmer Life Sciences (Boston, MA, USA). Acetyl coenzyme A sodium salt was purchased from Sigma Chemical Co. (St. Louis, MO, USA), and tryptamine–HCl was from Serva (Heidelberg, Germany). SCH 23390 and MK 801 were purchased from Tocris Cookson Ltd. (Bristol, UK).

Statistical analysis

Data are expressed as the mean ± standard error of mean (SEM) values and were analyzed for statistical significance by one-way ANOVA followed by *post hoc* Student-Newman-Keuls test using Instat Software (version 3.05 for Windows 95, GraphPad, San Diego, USA).

Results

In pineal glands of chicks kept under DD conditions AANAT activity fluctuated with a robust circadian rhythm, with low values during the subjective light phase (CT0-CT12) and high values during the subjective dark phase (CT12-CT24) of the cycle (Fig. 2, 3). One hour exposure of chicks to UV-A radiation or to a low intensity (4 lux) white light, acting on the eyes only, during the second half of the subjective dark phase (CT21-CT22) potently decreased AANAT activity in the pineal gland (Fig. 1). In line with our earlier observations [9, 25], the suppressive effect of retinally perceived UV-A light on pineal AANAT activity was antagonized by MK 801 (a selective blocker of NMDA type of glutamate receptors), injected into both eyes at a dose of 3 nmol/eye, and not affected by SCH 23390 (100 nmol/eye), a selective antagonist of D1-dopamine receptors (Fig. 1A). On the other hand, the decline of pineal AANAT activity produced by

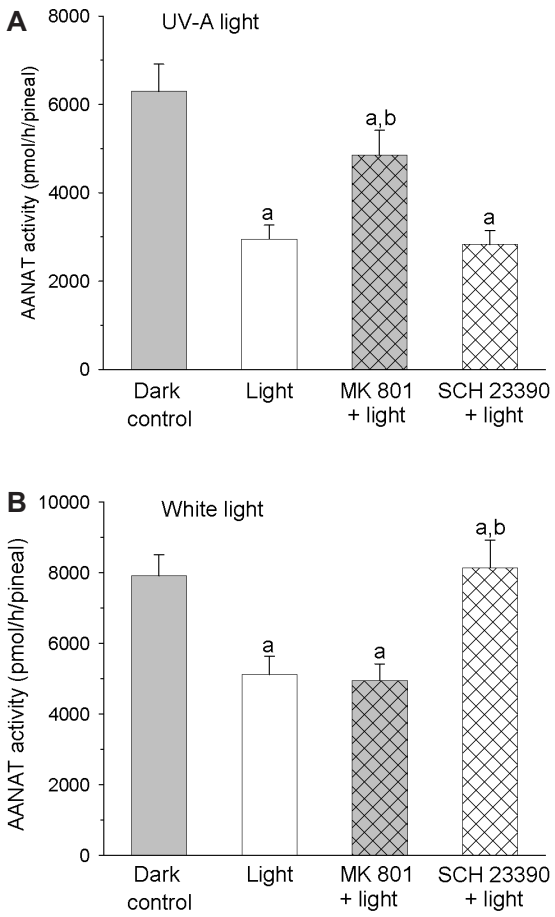

Fig. 1. Effects of intraocular administration of MK 801 (3 nmol/eye) and SCH 23390 (100 nmol/eye) on the suppressive action of retinal illumination with UV-A **(A)** and white light **(B)** on the nighttime AANAT activity in the chick pineal gland. For experimental details see text. Values are the means ± SEM (n = 5/group). [a] $p < 0.05$ *vs.* control, [b] $p < 0.05$ *vs.* light-treatment

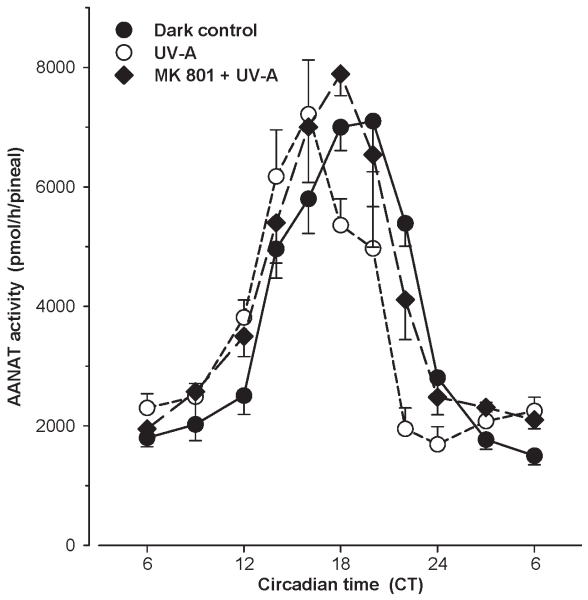

Fig. 2. MK 801 (3 nmol/eye, injected *ioc* into both eyes) attenuated the phase-advancing effect of retinal illumination with UV-A on the circadian rhythm of AANAT activity in the chick pineal gland. For experimental details see text. CT12 and CT24 refer, respectively, to the beginning and end of the subjective dark phase. Values are the means ± SEM (n = 5–6/group)

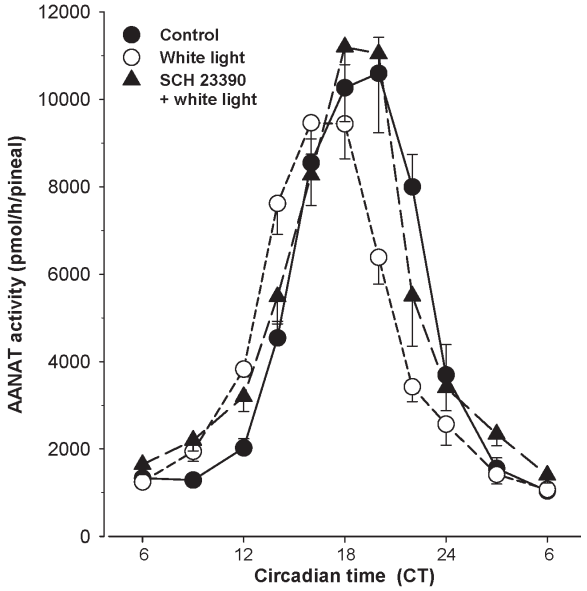

Fig. 3. SCH 23390 (100 nmol/eye, injected *ioc* into both eyes) attenuated the phase-advancing effect of retinal illumination with white light on the circadian rhythm of AANAT activity in the chick pineal gland. For experimental details see text. CT12 and CT24 refer, respectively, to the beginning and end of the subjective dark phase. Values are the means ± SEM (n = 5–6/group)

retinally perceived white light was blocked by SCH 23390, and not modified by MK 801 (Fig. 1B).

Ocular exposure of chicks to UV-A radiation or to white light produced a significant phase advance of the circadian rhythm of pineal AANAT activity compared to non-exposed controls, visible on a subsequent day of DD (Fig. 2, 3). The phase-shifting effect of retinally perceived UV-A pulse was attenuated by pretreatment of birds with MK 801 (3 nmol/eye) (Fig. 2). On the other hand, *ioc* injection of SCH 23390 (100 nmol/eye) blocked the phase shifting action of retinally perceived white light on the pineal AANAT activity rhythm (Fig. 3). Neither MK 801 nor SCH 23390 modified the circadian rhythm in AANAT activity in the chick pineal (results not shown).

Discussion

Accumulating spectrophotometric, electrophysiological, behavioral, and molecular data indicate that sensitivity to UV light is a general property of most classes of vertebrates, including teleost fish, amphibians, reptiles, birds, and some mammals [16, 27]. Many vertebrates use UV vision for such basic behaviors as foraging, social signaling and communication, mating, and migration/orientation [6, 16]. In studies performed on rodents it has been demonstrated that UV-A radiation (with wavelengths in the range of 320–400 nm), like visible light, blocks the short photoperiod-induced regression of the reproductive system, entrains circadian rhythm of locomotor activity and temperature, and induces robust expression of the transcription factor Fos in the suprachismatic nuclei and visual cortex [2, 3, 10]. It has also been shown that exposure of rodents and birds (chickens) to UV-A light at night dramatically decreases melatonin synthesis in the pineal gland and retina [8, 10, 11, 25, 36–38]. The suppressive effect of UV-A radiation on AANAT activity and melatonin content in the chick pineal gland was evoked by light acting directly on the gland [26]. In addition, UV-A radiation acting indirectly through the retina activates a cascade of yet poorly identified processes leading to suppression of melatonin synthesis in the pineal gland [25]. Results of the current study have demonstrated, for the first time, that retinal illumination with UV-A light provides a powerful and important signal that phase shifts

the circadian oscillator generating rhythmic activity in the chick pineal gland. The direction and magnitude of the UV-A-evoked changes in the circadian rhythm of pineal AANAT activity were comparable to these produced by the low intensity (4 lux) white light.

Molecular and neurochemical components of the multisynaptic pathway conveying the light information from the chick retina to the pineal gland remain to be elucidated. At the level of the retina, signals triggered by the absorption of UV-A and white light appear to involve different neurotransmitter systems. Both the acute suppressive and phase-shifting effects of UV-A radiation was mediated, in part, by retinal NMDA glutamate receptors [9, 25; present results], whereas the actions of white light was dependent on the activation of D1-dopamine receptors [9, 35; present results]. On the other hand, it has been demonstrated that regulation of melatonin synthesis in the chicken pineal gland by retinally perceived UV-A or white light does not depend on stimulation of kainate/AMPA glutamate and D2-dopamine receptors, respectively [18, 25, 35]. An involvement of different classes of receptors in the actions of UV-A and white light suggests that different photoreceptor cells containing different photopigments may participate in the responses to these distinct radiation types.

Classical photoreceptors, cones and rods, contain photopigments from the opsin family. In the chicken four cone visual pigments with the maximum absorbance (λ_{max}) at about 415 nm (violet/ultraviolet sensitive; VS/UVS), 455 nm (short wave sensitive; SWS), 505 nm (middle wave sensitive; MWS), 569 nm (long wave sensitive; LWS), and one rod pigment (λ_{max} = 506 nm) have been identified. Genes encoding these pigments have been isolated, sequenced and expressed [16]. The range of spectral sensitivity of VS/UVS cones together with a postulated role of glutamate as the crucial neurotransmitter involved in photic entrainment [15] provides a basis to hypothesize that in the chicken retina absorption of UV-A light by this class of cones could trigger a cascade of biochemical events that are subsequently sent downstream to the pineal gland and regulate its activity.

Experimental data accumulated during the last decade indicate that, in addition to the classical photoreceptors, retinas of various vertebrates, including chicken, contain a novel class of photoreceptive cells. Those cells contain melanopsin, a newly discovered photopigment (λ_{max} around 480 nm), and have been shown to provide information regarding environmental irradiance for a variety of non-image forming light responses, including circadian entrainment (reviewed by [22, 32]). Two types of melanopsin, Opn4x and Opn4m, have recently been discovered and characterized in the chicken [7, 12, 29]. In the retina of mammals melanopsin is present in a subpopulation of ganglion cells (intrinsically photoreceptive retinal ganglion cells; ipRGCs). Vast majority of ipRGCs is located in the ganglion cell layer; a sparse population of ipRGCs is displaced into the inner nuclear layer [13, 31, 32]. In the chicken retina melanopsin mRNA expression was observed primarily in the inner nuclear layer (most likely in horizontal and bipolar cells) and, to a lesser extent, in ganglion cells [12, 29]. It appears likely that melanopsin-containing cells in the inner nuclear layer of the chicken retina may influence the activity of adjacent inner retinal neurons, including dopaminergic ones. In line with this hypothesis is the recent report on direct contacts between melanopsin-positive cells and dopaminergic amacrine cells in the rat and human retina [31]. In which way melanopsin-containing cells and dopaminergic cells communicate in the chicken retina and what is a functional significance of this communication is at present unknown.

Acknowledgments:
This work was supported by the grant from the Medical University of Łódź, Poland (No. 502-13-409). The authors thank Professor J.Z. Nowak for fruitful discussion. The skillful assistance of Dr. Jolanta Rosiak and Mrs Teresa Kwapisz is highly appreciated.

References:

1. Allen AE, Pang SF, Nir I: The effect of environmental photoperiodicity on indole rhythms and locomotor activity in sighted and eye covered chickens. J Neural Transm Gen Sect, 1991, 83, 107–119.
2. Amir S, Robinson B: Ultraviolet light entrains rodent suprachiasmatic nuclei pacemaker. Neuroscience, 1995, 69, 1005–1011.
3. Amir S, Robinson B: Fos expression in rat visual cortex induced by ocular input of ultraviolet light. Brain Res, 1996, 716, 213–218.
4. Arendt J (Ed.): Melatonin and the Mammalian Pineal Gland. Chapman and Hall, London, 1995.
5. Barrett RK, Underwood H: Retinally perceived light can entrain the pineal melatonin rhythm in Japanese quail. Brain Res, 1991, 563, 87–93.
6. Bennett ATD, Cuthill KC: Ultraviolet vision in birds: what is its function? Vis Res, 1994, 34, 1471–1478.
7. Bellingham J, Chaurasia S, Melyan Z, Liu C, Cameron MA, Tarttelin EE, Iuvone PM et al.: Evolution of melanopsin photoreceptors: discovery and characterization of

a new melanopsin in nonmammalian vertebrates. PLoS Biol, 2006, 4, 1334–1342.
8. Benshoff HM, Brainard GC, Rollag MD, Lynch GR: Suppression of pineal melatonin in *Peromyscus leucopus* by different monochromatic wavelengths of visible and near-ultraviolet light (UV-A). Brain Res, 1987, 420, 391–402.
9. Berezińska M, Lorenc A, Rosiak J, Zawilska JB: Photic control of pineal melatonin synthesis: an input from different photoreceptors. Pharmacol Rep, 2006, 58, 327.
10. Brainard GC, Barker FM, Hoffman RJ, Stetson MH, Hanifin JP, Podolin PL, Rollag MD: Ultraviolet regulation of neuroendocrine and circadian physiology in rodents. Vis Res, 1994, 32, 521–532.
11. Brainard GC, Podolin PL, Leivy SW, Rollag MD, Cole C, Barker FM: Near-ultraviolet radiation suppresses pineal melatonin content. Endocrinology, 1986, 119, 2201–2205.
12. Chaurasia SS, Rollag MD, Jiang G, Hayes WP, Haque R, Natesan A, Zatz M et al.: Molecular cloning, localization and circadian expression of chicken melanopsin (*Opn4*): differential regulation of expression in pineal and retinal cell types. J Neurochem, 2005, 92, 158–170.
13. Dacey DM, Liao HW, Peterson BB, Robinson FR, Smith VC, Pokorny J, Yau KW, Gamlin PD: Melanopsin-expressing ganglion cells in primate retina signal colour and irradiance and project to the LGN. Nature, 2005, 433, 749–754.
14. Ganguly S, Coon SL, Klein DC: Control of melatonin synthesis in the mammalian pineal gland: the critical role of serotonin acetylation. Cell Tiss Res, 2002, 309, 127–137.
15. Hannibal J: Neurotransmitters of the retino-hypothalamic tract. Cell Tiss Res, 2002, 309, 73–88.
16. Hunt DM, Wilkie SE, Bowmaker JK, Poopalasundaram S: Vision in the ultraviolet. Cell Mol Life Sci, 2001, 58, 1583–1598.
17. Iuvone PM, Tosini G, Pozdeyev N, Haque R, Klein DC, Chaurasia SS: Circadian clocks, clock networks, arylalkylamine N-acetyltransferase, and melatonin in the retina. Prog Retin Eye Res, 2005, 24, 433–456.
18. Morgan IG, Boelen MK, Miethke T: Pineal activity is under the control of retinal D1-dopaminergic pathways. Neuroreport, 1995, 6, 446–448.
19. Morgan IG, Boelen MK, Miethke T: Parallel suppression of retinal and pineal melatonin synthesis by retinally mediated light. Neuroreport, 1995, 6, 1530–1532.
20. Nakahara K, Murakami N, Nasu T, Kuroda H, Murakami T: Individual pineal cells of chick possess photoreceptive, circadian clock and melatonin-synthesizing capacities in vitro. Brain Res, 1997, 774, 242–245.
21. Natesan A, Geetha L, Zatz M: Rhythm and soul in the avian pineal. Cell Tiss Res, 2002, 309, 35–45.
22. Nayak SK, Jegla T, Panda S: Role of a novel photopigment, melanopsin, in behavioral adaptation to light. Cell Mol Life Sci, 2007, 64, 144–154.
23. Nowak JZ, Żurawska E, Zawilska J: Melatonin and its generating system in vertebrate retina: circadian rhythm, effect of environmental lighting and interaction with dopamine. Neurochem Int, 1989, 14, 397–406.
24. Pittendrigh CS: Circadian systems: entrainment. In: Handbook of Behavioral Neurobiology, Biological Rhythms, 4th edn. Ed. Aschoff J, Plenum Press, NY, 1981, 57–80.
25. Rosiak J, Zawilska JB: Near-ultraviolet light perceived by the retina generates the signal suppressing melatonin synthesis in the chick pineal gland – an involvement of NMDA receptors. Neurosci Lett, 2005, 379, 214–217.
26. Rosiak J, Iuvone PM, Zawilska JB: UV-A light regulation of arylalkylamine N-acetyltransferase activity in the chick pineal gland: role of cAMP and proteasomal proteolysis. J Pineal Res, 2005, 39, 419–424.
27. Shi Y, Radlwimmer B, Yokoyama S: Molecular genetics and the evolution of ultraviolet vision in vertebrates. Proc Natl Acad Sci USA, 2001, 98, 11731–11736.
28. Takahashi JS, Murakami N, Nikaido SS, Pratt BL, Robertson LM: The avian pineal gland, a vertebrate model system of the circadian oscillator: cellular regulation of circadian rhythms by light, second messengers, and macromolecular synthesis. Recent Prog Hormone Res, 1989, 45, 279–352.
29. Tomonari S, Takagi A, Akamatsu S, Noji S, Ohuchi H: A non-canonical photopigment, melanopsin, is expressed in the differentiating ganglion, horizontal, and bipolar cells of the chicken retina. Dev Dynam, 2005, 234, 783–790.
30. Underwood H, Steele CT, Zivkovic BD: Circadian organization and the role of the pineal gland in birds. Microsc Res Tech, 2001, 53, 48–62.
31. Vugler AA, Redgrave P, Semo M, Lawrence J, Greenwood J, Coffey PJ: Dopamine neurons form a discrete plexus with melanopsin cells in normal and degenerating retina. Exp Neurol, 2007, 205, 26–35.
32. Zawilska JB, Czarnecka K: Melanopsin: a newly discovered photoreceptor controlling circadian rhythms. Adv Cell Biol, 2006, 33, 229–246.
33. Zawilska JB, Nowak JZ: Melatonin: from biochemistry to therapeutic applications. Pol J Pharmacol, 1999, 51, 3–23.
34. Zawilska JB, Berezińska M, Lorenc A, Skene DJ, Nowak JZ: Retinal illumination phase-shifts the circadian rhythm of serotonin *N*-acetyltransferase activity in the chicken pineal gland. Neurosci Lett, 2004, 360, 153–155.
35. Zawilska JB, Berezińska M, Rosiak J, Skene DJ, Vivien-Roels B, Nowak JZ: Suppression of melatonin biosynthesis in the chicken pineal gland by retinally perceived light – involvement of D1-dopamine receptors. J Pineal Res, 2004, 36, 80–86.
36. Zawilska JB, Rosiak J, Nowak JZ: Effects of near-ultraviolet light on the nocturnal serotonin *N*-acetyltransferase activity of rat pineal gland. Neurosci Lett, 1998, 243, 48–52.
37. Zawilska JB, Rosiak J, Nowak JZ: Near-ultraviolet radiation suppresses melatonin synthesis in the chicken retina. A role of dopamine. Life Sci, 2000, 67, 2233–2246.
38. Zawilska JB, Rosiak J, Trzepizur K, Nowak JZ: The effects of near-ultraviolet light on serotonin N-acetyltransferase activity in the chick pineal gland. J Pineal Res, 1999, 26, 122–127.

Received:
April 17, 2007; in revised form: August 20, 2007.