Effects on hypothalamus when CPG is fed back to basal ganglia based on KIV model

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Abstract The KIV model approximates the operation of the basic vertebrate forebrain together with the basal ganglia and motor systems. In KIV model, the hypothalamus and the basal ganglia which are two important parts in the midline forebrain are closely associated with the locomotion. The CPG model with time delay is established in this paper and the stability of this CPG model is discussed. The CPG output is treated as the proprioception and fed back to the basal ganglia. We focus on the effects on the hypothalamus and the basal ganglia when the time delay parameter a_d , the CPG amplitude parameter e and the CPG frequency parameter T_r are changed. Through analysis, we find that there exists optimum value of the parameters a_d or T_r which can make the synchronization of the hypothalamus optimum when the CPG is added into the basal ganglia. The results could have important implications for biological processes which are about interaction between the neural network and the CPG.

Keywords Central pattern generator · KIV model · Time delay · Dynamic characteristics

Introduction

Locomotion is a purposeful, goal-directed behavior initiated by signals arising from either volitional processing in the cerebral cortex or emotional processing in the limbic system and locomotion is controlled by neural structure (Grillner

2011; Takakusaki 2013). Neurobiological studies revealed that rhythmic motor patterns are controlled by neural oscillators, referred to as the CPG, which generate oscillatory signals (Jellies and Kueh 2012; Cheron et al. 2012). Many researchers have studied the interaction between the neural system and the CPG (Harris-Warrick 2011; Taga 1998; Minassian et al. 2012; Lu and Tian 2014).

The KIV model is an intentional dynamical system, in which meaningful knowledge is continuously created, utilized, and dissipated in the form of sequences of oscillatory patterns of activity modulated by simulated synaptic connections previously modified by learning sensory data (Kozma and Freeman 2003, 2009; Kozma et al. 2003). In KIV model, a KIII model is used to simulate the midline forebrain which receives the inter-ceptor signals through the basal ganglia and processes them in the hypothalamus and the septum (Kozma et al. 2003). The interoceptors includes proprioception from the musculoskeletal system and interoception from the vital organs including the cardiovascular and respiratory systems (Kozma and Freeman 2009). In this model, the basal ganglia refers to a group of subcortical nuclei that includes the striatum, globus pallidus and connected structures (Visser and Bloem 2005). The basal ganglia is involved in the control of voluntary movements (Middleton and Strick 2000) and contributes to the planning and execution of voluntary movements via a series of parallel basal ganglia thalamocortical loops (Takakusaki et al. 2004). The basal ganglia outflow is also directed to the motor networks in the brainstem where fundamental neuronal networks for controlling muscle tone and locomotor movements are located (Takakusaki et al. 2003, 2004). The hypothalamus is also involved in the locomotion (Saper et al. 2005; Krout et al. 2003). In this paper, we focus on the dynamic characteristics of the

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basal ganglia and the hypothalamus when the output of CPG is fed back to the basal ganglia.

The proprioception is the ability to know where our body is at all times. Kondo and Ito (2005) showed that the automatic movement was mainly realized at the spinal cord based on proprioceptive feed-back. Overholt et al. (2002) treated the proprioception as a sense of body position and movement that supported the control over many automatic motor functions such as posture and locomotion. They demonstrated that the proprioception provided the central nervous system with the information about the spatial location of body parts. The central nervous system needs to know the angles of all joints between the hand and the body so as to combine these angles with the known segment lengths to localize a hand. Bartsch and Kirchner (2006) treated the CPG as a proprioception in the locomotion of the humanoid robot. In this paper, the CPG is treated as the proprioception from the musculoskeletal system and is fed back to the basal ganglia. The researchers have already revealed that time delays can gradually affect the spatio-temporal dynamics in networks of coupled neurons (Roxin et al. 2005). Therefore, when the CPG signal is sent to the basal ganglia, the time delay should be considered.

This paper is organized as follows. In “[The model between the new CPG model and the midline forebrain](#)” section, a new CPG model with time delay is built, and we discuss the stability of the new model. Then the model between the new CPG and the midline forebrain (MF) is established. Detailed analysis for effects on the basal ganglia and the hypothalamus when the CPG’s parameters are changed is shown in “[Analyses on parameters’ effects](#)” section. The conclusions and future works are made in “[Conclusions](#)” section.

The model between the new CPG model and the midline forebrain

The CPG model with time delay

The impulse response function of time delay $h_d(t)$ (Huang et al. 2011) can be used as

$$h_d(t) = \begin{cases} Aa_d t e^{-a_d t} & (t \geq 0) \\ 0 & (t < 0) \end{cases}$$

The parameter a_d lumps the characteristic delays of the synaptic transmission together. If the input is $x(t)$, the output is $y(t) = h_d * x(t)$.

This operator can be described as a second-order ordinary differential equation

$$\ddot{y}(t) = Aa_d x(t) - 2a_d \dot{y}(t) - a_d^2 y(t).$$

And it can be written as follows:

$$\begin{cases} \dot{y}(t) = z(t) \\ \dot{z}(t) = Aa_d x(t) - 2a_d z(t) - a_d^2 y(t) \end{cases} \quad (1)$$

Choosing the CPG output (Matsuoka 2011) as the input of Eq. 1, the state equation of the new CPG model is given by

$$\begin{cases} \dot{x}_1 = x_2 \\ \dot{x}_2 = Aa_d \max(0, x_3) - 2a_d x_2 - a_d^2 x_1 \\ T_r \dot{x}_3 + x_3 = -dx_4 + e \\ T_a \dot{x}_4 + x_4 = \max(0, x_3). \end{cases} \quad (2)$$

The linear stability analysis is in “[Appendix](#)” section. According to the result from analysis, system (2) is stable.

The KIV model

Higher cognition is modeled here using KIV set. Kozma et al. (2003) showed the structure of a KIV model. Three types of sensory signals can be distinguished. Each of these sensory signals provides stimulus to a given part of the brain, namely the sensory cortices, midline forebrain, and the hippocampal formation, respectively.

The corresponding types of sensory signals are listed below: (1) exteroceptors; (2) interoceptors, including proprioception from the musculoskeletal system and interoception from the vital organs including the cardiovascular and respiratory systems; (3) orientation signals. The convergence location and output are provided by the amygdala.

In KIV model, the MF receives the interoceptor signals through the basal ganglia and processes them in the hypothalamus and the septum. The MF provides the value system of the KIV, using information on the internal goals and conditions in the animal. It provides the “Why?” stream to the amygdala, which combines this with the “What?” and “Where?” information coming from the cortex and the hippocampus to make a decision about the next step/action to be taken.

The basic K-unit, called K0 set, models a neuron population of about 10^4 neurons. Its dynamics are governed by a second-order ordinary differential equation (Kozma et al. 2003):

$$(a * b) \frac{d^2 P(t)}{dt^2} + (a + b) \frac{dP(t)}{dt} + P(t) = F(t)$$

Here a and b are biologically determined time constants; $a = 0.22$, $b = 0.72$. $P(t)$ denotes the activation of the node as function of time; $F(t)$ is the summed activation from neighbor nodes and any external input. The K0 set has a weighted input and an asymptotic sigmoid

Fig. 1 Model between the CPG and the MF

function for the output. The sigmoid function $Q(x)$ is given by the equation:

$$Q(x) = q * \left\{ 1 - \exp \left[-\frac{1}{q(e^x - 1)} \right] \right\}$$

where $q = 5$, is the parameter specifying the slope and maximal asymptote of the curve. Coupling two or more K0 sets with excitatory connections, we get a KI. The next step in the hierarchy is the KII model. KII is a double-layer of excitatory and inhibitory units. In the simplest architecture there are 4 nodes: two excitatory, denoted e , and two inhibitory, denoted i , nodes. The excitatory and inhibitory nodes in a KII set are arranged in corresponding layers, so KII has a double-layer structure. Given proper initial conditions, this model may produce sustained periodic oscillations the frequency and magnitude of which are determined by the interconnection weights between units. KIII consists of three double layers of KII sets that are connected with no-delay feed-forward connections and delayed feed-back connections. Properly tuned KIII models typically exhibit non-convergent chaotic behavior due to the competition of KII oscillators. Finally, several KIII and/or KII sets form the multi-sensory KIV set. KIV is capable of exhibiting intermittent spatio-temporal synchronization, as the result of interacting chaotic KIII and KII oscillatory units. In the following, the KIV model is formed of 64 channels. The state equations of the KIV model and the values of these parameters are in (Fu 2009).

The model between the CPG model and the MF based on KIV set

The MF is treated as the KIII model in KIV set. The hypothalamus (HT), diagonal band (DB) and septum are models as KII units, while basal ganglia (BC) is a KI unit. The MF receives the proprioception (CPG) signals through the basal ganglia and processes them in the hypothalamus and the septum (Kozma et al. 2003). Then the model between the new CPG model and the MF based on KIV set is shown by (Fig. 1).

Analyses on parameters' effects

The basal ganglia plays important roles in the regulation of movements (Takakusaki and Okumura 2008). The CPG with time delay is treated as the proprioception from the musculoskeletal system and is fed back to the basal ganglia and we focus on the dynamic characteristics. The relationship between the midline forebrain and the CPG is studied in two aspects. One is the effects on the midline forebrain when the parameter a_d of time delay is varied. The other one is effects on the midline forebrain when three parameters T_r and e are varied. The frequency of the limit cycle oscillation in the CPG is proportional to $1/T_r$ and the amplitude is proportional to the parameter e (Matsuoka 2011). In the following these parameters are set as $A = 3.25$, $T_r = 0.1$, $T_a = 2T_r$, $d = 2.5$ and $e = 8$.

The effects on the basal ganglia and the hypothalamus with parameter a_d

A useful diagnostic of phase synchronization is the mean field of the ensemble and it is defined as

$$X_n = \frac{1}{N} \sum_{i=1}^N x_i \quad (3)$$

where N is the number in the ensemble.

A quantitative measure of synchronization is the variance of mean field oscillation. The synchronization becomes stronger when the value of the variable $Var(X)$ is close to 1. When $Var(X) = 1$, the state of all neurons is complete synchronization. The $Var(X)$ is given by the equation:

Fig. 2 $Var(X)$ of the hypothalamus output G_{i1} with different a_d

$$Var = \frac{1}{M-1} \sum_{i=1}^M \left(X_i - \frac{1}{M} \sum_{j=1}^M X_j \right)^2$$

where M is the number of the samples and X_i is normalized.

Then we calculate the $Var(X)$ with different a_d and x_i in Eq. 3 is replaced by the hypothalamus output G_{i1} in the KIV model. In following simulation, the time interval is [1, 2,000 ms] and the CPG signals are sent to the basal ganglia in the interval [200, 800 ms]. The plots are exhibited in Figs. 2, 3. In Fig. 2, the delay time a_d is varied in the interval [1, 20].

From Fig. 2, we can see that there exists an optimum value of a_d which is 8 and it makes the synchronization best. In Fig. 3, the amplitude of G_{i1} are the biggest when $a_d = 8$.

Now we analyze the systematic error, careless error and limit error of the values in Fig. 2.

According to the Bessel's formula and the Peters' formula which are shown in Eqs. 4 and 5 (Fei 2004), the standard deviation σ and σ' are obtained

$$\sigma = \sqrt{\frac{\sum_{i=1}^n \left(x_i - \frac{1}{n} \sum_{j=1}^n x_j \right)^2}{n-1}} \quad (4)$$

$$\sigma' = 1.253 \frac{\sum_{i=1}^n \left| x_i - \frac{1}{n} \sum_{j=1}^n x_j \right|}{\sqrt{n(n-1)}} \quad (5)$$

The value of $|u|$ is shown as follows

$$|u| < \frac{2}{\sqrt{n-1}} = \frac{2}{\sqrt{8}} \approx 0.7072$$

where $n = 9$, $u = \frac{\sigma'}{\sigma} - 1 = 1.0803 - 1 = 0.0803$.

Therefore, the values in Fig. 2 have no systematic error.

Fig. 3 G_{i1} and G_{641} with different a_d . **a** $a_d = 1$, **b** $a_d = 8$, **c** $a_d = 16$

The maximum value of the values in Fig. 2 is 0.2485 and the minimum one is 0.2349.

Based on Eq. 6, the values of $g(\min i)$ and $g(\max i)$ are 1.6647 and 1.2391.

$$g(i) = \frac{\left| x_i - \frac{1}{n} \sum_{j=1}^N x_j \right|}{\sigma} \quad (6)$$

According to the critical value table (Fei 2004), the value of $g_0(n, \alpha)$ is obtained

$$g_0(n, \alpha) = g_0(9, 0.05) = 2.11.$$

Because the values of $g(\min i)$ and $g(\max i)$ are both lower than 2.11, the values in Fig. 2 have no careless error.

According to the t distribution table (Fei 2004), the value of t_α is 2.31. The limit error is shown as follows

$$\delta_{\lim} = \pm t_\alpha \cdot \sigma_{\bar{x}} = \pm 0.0036$$

where $\alpha = 0.05$ and $\sigma_{\bar{x}} = \frac{\sigma}{\sqrt{n}} = 0.0016$.

The effects on the basal ganglia and the hypothalamus with parameters T_r and e .

Now we calculate the $\text{Var}(X)$ with different values of T_r and e . The plots are exhibited in Figs. 4, 5, and 6. In Fig. 4, the parameter T_r is varied in the interval $[0.1, 0.9]$. In Fig. 6, the parameter e is varied in the interval $[1, 30]$.

From Fig. 4, we can see that there exists an optimum value of T_r which is 0.6 and it makes the synchronization of the hypothalamus output best. In Fig. 5, the amplitude of

Fig. 4 $\text{Var}(X)$ of the hypothalamus output G_{11} with different T_r

Fig. 6 $\text{Var}(X)$ of the hypothalamus output G_{11} with different e

Fig. 5 G_{11} and G_{641} with different T_r . **a** $T_r = 0.05$, **b** $T_r = 0.6$, **c** $T_r = 0.8$

G_{i1} are the biggest when $T_r = 0.6$. However, the synchronization value increases with the increasing of the parameter e in Fig. 6.

Then we analyze the systematic error, careless error and limit error of the values in Fig. 4.

According to Eqs. 4 and 5, the value of $|u|$ is shown as follows

$$|u| < \frac{2}{\sqrt{n-1}} = \frac{2}{\sqrt{8}} \approx 0.7072$$

where $n = 9$, $u = \frac{\sigma'}{\sigma} - 1 = 1.0465 - 1 = 0.0465$.

Therefore, the values in Fig. 4 have no systematic error.

The maximum value of the values in Fig. 4 is 0.2170 and the minimum one is 0.0474. Based on Eq. 6, the values of $g(\min i)$ and $g(\max i)$ are 1.1925 and 1.7870. Because the values of $g(\min i)$ and $g(\max i)$ are both lower than 2.11, the values in Fig. 4 have no careless error.

The limit error is shown as follows

$$\delta_{\lim} = \pm t_{\alpha} \quad \sigma_{\bar{x}} = \pm 0.0438$$

where $\sigma_{\bar{x}} = 0.0190$.

In the following, we analyze the systematic error, careless error and limit error of the values in Fig. 6.

According to Eqs. 4 and 5, the value of $|u|$ is shown as follows

$$|u| < \frac{2}{\sqrt{n-1}} = \frac{2}{\sqrt{8}} \approx 0.7072$$

where $n = 9$, $u = \frac{\sigma'}{\sigma} - 1 = 1.0461 - 1 = 0.0461$.

Therefore, the values in Fig. 6 have no systematic error.

The maximum value of the values in Fig. 6 is 0.1643 and the minimum one is 0.0365. Based on Eq. 6, the values of $g(\min i)$ and $g(\max i)$ are 1.1727 and 1.6473. Because the values of $g(\min i)$ and $g(\max i)$ are both lower than 2.11, the values in Fig. 6 have no careless error.

The limit error is shown as follows.

$$\delta_{\lim} = \pm t_{\alpha} \quad \sigma_{\bar{x}} = \pm 0.0349$$

where $\sigma_{\bar{x}} = 0.0151$.

Conclusions

In this paper, The CPG model with time delay is established and the stability of this CPG model is discussed. The CPG output is treated as the proprioception and fed back to the basal ganglia. We analyze the effects on the hypothalamus and the basal ganglia when the parameters a_d , e and T_r are changed.

From analysis, we can see that the synchronization of the hypothalamus depends on the structure of the CPG, such as the time delay parameter a_d , the CPG frequency parameter T_r and the amplitude parameter e . There exists

Fig. 7 Phase diagram when $T_r = 0.6$, $a_d = 8$ and $e = 4$

an optimal value of T_r or a_d at which the synchronization and the amplitude get their peak value.

According to Skarda and Freeman (1987), the normal background activity of neural systems is a chaotic state. In the perceptual systems, input from the sensors perturbs the neuronal ensembles from the chaotic background. The result is that the system transitions into a new attractor that represents the meaning of the sensory input, given the context of the state of the organism and its environment (Harter and Kozma 2005). The phase diagram of M_{11} and G_{11} is chaotic when no signal is fed back to the basal ganglia. When $T_r = 0.6$, $a_d = 8$ and $e = 4$, the phase diagram is obtained in Fig. 7.

From Fig. 7, we can see that the hypothalamus transitions into a new attractor that represents the meaning of the CPG input. Considering the relationship between the brain and the locomotion, the results could have important implications for many biological processes which are about the neural network.

The K-sets have been used for dynamic memory designs and for robust classification and pattern recognition (Gutierrez-Osuna and Gutierrez-Galvez 2003; Harter and Kozma 2005). It is deserved further investigation that the K-sets are used for the classification and pattern recognition of the CPG which corresponds to different locomotion (Dong et al. 2011).

Appendix

In order to analyze dynamic performance of the new CPG model, let's consider the following two cases for system (2) and we obtain the fixed points as

$$\begin{cases} (a) \left[\frac{Ae}{a_d(1+d)}, 0, \frac{e}{1+d}, \frac{e}{1+d} \right], & x_3 > 0 \\ (b) [0, 0, e, 0], & x_3 \leq 0 \end{cases}$$

We linearize it and calculate its Jacobian matrix, i.e. the partial derivative J at the fixed point. At the fixed point, we have

$$J_1 = \begin{bmatrix} 0 & 1 & 0 & 0 \\ -a_d^2 & -2a_d & Aa_d & 0 \\ 0 & 0 & -1/T_r & -d/T_r \\ 0 & 0 & 1/T_a & -1/T_a \end{bmatrix} (x_3 > 0),$$

$$J_2 = \begin{bmatrix} 0 & 1 & 0 & 0 \\ -a_d^2 & -2a_d & 0 & 0 \\ 0 & 0 & -1/T_r & -d/T_r \\ 0 & 0 & 0 & -1/T_a \end{bmatrix} (x_3 \leq 0)$$

when $x_3 > 0$, the characteristic equation is described by

$$\det(J_1 - \lambda I) = \begin{bmatrix} -\lambda & 1 & 0 & 0 \\ -a_d^2 & -2a_d - \lambda & Aa_d & 0 \\ 0 & 0 & (-1/T_r) - \lambda & -d/T_r \\ 0 & 0 & 1/T_a & (-1/T_a) - \lambda \end{bmatrix}.$$

Eigenvalues are given by

$$\begin{aligned} \lambda_1 &= \lambda_2 = -a_d, \lambda_{3,4} \\ &= \frac{-(T_a + T_r) \pm \sqrt{(T_a + T_r)^2 - 4T_a T_r(1+d)}}{2T_a T_r} \\ &= \frac{-(T_a + T_r) \pm \sqrt{(T_a - T_r)^2 - 4dT_a T_r}}{2T_a T_r}. \end{aligned}$$

Parameters T_r and T_a are the time constants that determine the reaction times of variables x_3 and x_4 , respectively. Then the values of T_r and T_a are both positive. The parameter d is a coefficient of the adaptation effect and the value is positive (Matsuoka 1985, 2011). The value of the parameter a_d is also positive. Hence, the value of λ_1 and λ_2 are all negative. The real part of λ_3 and λ_4 are also negative. Therefore, system (2) is stable.

When $x_3 \leq 0$, the characteristic equation is described by

$$\det(J_2 - \lambda I) = \begin{bmatrix} -\lambda & 1 & 0 & 0 \\ -a_d^2 & -2a_d - \lambda & 0 & 0 \\ 0 & 0 & (-1/T_r) - \lambda & -d/T_r \\ 0 & 0 & 0 & (-1/T_a) - \lambda \end{bmatrix}.$$

Eigenvalues are given by

$$\lambda_1 = \lambda_2 = -a_d, \lambda_3 = \frac{-1}{T_r}, \lambda_4 = \frac{-1}{T_a}.$$

Because the values of these four eigenvalues are all negative, system (2) is stable.

References

- Bartsch S, Kirchner F (2006) Robust control of a humanoid robot using a bio-inspired approach based on central pattern generators, reflexes, and proprioceptive feedback. In: IEEE international conference on robotics and biomimetics. Kunming, China, pp 1547–1552
- Cheron G, Duvinage M, Saedeleer CD et al (2012) From spinal central pattern generators to cortical network: integrated BCI for walking rehabilitation. *Neural Plast* 2012:375148
- Dong W, Wang R, Zhang Z (2011) Exploring human rhythmic gait movement in the role of cerebral cortex signal. *Appl Math Mech Engl Ed* 32(2):223–230
- Fei YT (2004) Error theory and data processing. China Machine Press, Beijing, pp 10–56
- Fu J (2009) Bionic information processing technique for electronic nose based on an olfactory neural network, Dissertation. Zhejiang University
- Grillner S (2011) Human locomotor circuits conform. *Science* 334:912–913
- Gutierrez-Osuna R, Gutierrez-Galvez A (2003) Habituation in the KII olfactory model with chemical sensor arrays. *IEEE Trans Neural Netw* 14:1565–1568
- Harris-Warrick RM (2011) Neuromodulation and flexibility in Central Pattern Generator networks. *Curr Opin Neurobiol* 21(5):685–692
- Harter D, Kozma R (2005) Chaotic neurodynamics for autonomous agents. *IEEE Trans Neural Netw* 16(3):565–579
- Huang G, Zhang D, Meng J et al (2011) Interactions between two neural populations: a mechanism of chaos and oscillation in neural mass model. *Nerocomputing* 74:1026–1034
- Jellies J, Kueh D (2012) Centrally patterned rhythmic activity integrated by a peripheral circuit linking multiple oscillators. *J Comp Physiol A* 198(8):567–582
- Kondo T, Ito K (2005) Periodic motion control by modulating CPG parameters based on time-series recognition. In: The 8th European conference on artificial life. Canterbury, England, pp 906–915
- Kozma R, Freeman WJ (2003) Basic principles of the KIV model and its application to the navigation problem. *J Integr Neurosci* 2(1):125–146
- Kozma R, Freeman WJ (2009) The KIV model of intentional dynamics and decision making. *Neural Netw* 22(3):277–285
- Kozma R, Freeman WJ, Erdi P (2003) The KIV model-nonlinear spatio-temporal dynamics of the primordial vertebrate forebrain. *Neurocomputing* 52:819–826
- Krout KE, Mettenleiter TC, Loewy AD (2003) Single CNS neurons link both central motor and cardiosympathetic systems: a double-virus tracing study. *Neuroscience* 118:853–866S
- Lu Q, Tian J (2014) Synchronization and stochastic resonance of the small-world neural network based on the CPG. *Cogn Neurodyn* 8:217–226
- Matsuoka K (1985) Sustained oscillations generated by mutually inhibiting neurons with adaptation. *Biol Cybern* 52:367–376
- Matsuoka K (2011) Analysis of a neural oscillator. *Biol Cybern* 104:297–304
- Middleton FA, Strick PL (2000) Basal ganglia and cerebellar loops: motor and cognitive circuits. *Brain Res Rev* 31:236–250
- Minassian K, Hofstoetter U, Tansey K et al (2012) Neuromodulation of lower limb motor control in restorative neurology. *Clin Neurol Neurosurg* 114(5):489–497
- Overholt JL, Hudak GR, Gerhart GR (2002) Defining proprioceptive behaviors for autonomous mobile robots. In: Proceedings of SPIE, Orlando, FL, USA, pp 287–294

- Roxin A, Brunel N, Hansel D (2005) Role of delays in shaping spatiotemporal dynamics of neuronal activity in large networks. *Phys Rev Lett* 94:238103
- Saper CB, Lu J, Chou TC, Gooley J (2005) The hypothalamic integrator for circadian rhythms. *Trends Neurosci* 28(3):152–157
- Skarda CA, Freeman WJ (1987) How brains make chaos in order to make sense of the world. *Behav Brain Sci* 10:161–195
- Taga G (1998) A model of the neuro-musculo-skeletal system for anticipatory adjustment of human locomotion during obstacle avoidance. *Biol Cybern* 78(1):9–17
- Takakusaki K (2013) Neurophysiology of gait: from the spinal cord to the frontal lobe. *Mov Disord* 28(11):1483–1491
- Takakusaki K, Okumura T (2008) Neurobiological basis of controlling posture and locomotion. *Adv Robot* 22:1629–1663
- Takakusaki K, Habaguchi T, Ohtinata-Sugimoto J et al (2003) Basal ganglia efferents to the brainstem centers controlling postural muscle tone and locomotion: a new concept for understanding motor disorders in basal ganglia dysfunction. *Neuroscience* 119:293–308
- Takakusaki K, Saitoh K, Harada H, Kashiwayanagi M (2004) Role of basal ganglia-brainstem pathways in the control of motor behaviors. *Neurosci Res* 50:137–151
- Visser JE, Bloem BR (2005) Role of the basal ganglia in balance control. *Neural Plast* 12(2–3):161–174