Sensorimotor control of reactive balance in individuals with Parkinson's disease before and after adapted tango

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Postural instability in Parkinson’s disease leads to falls and injury

- PD is a neurodegenerative disorder affecting the basal ganglia in the brain

- Postural instability is a hallmark sign of PD

- 3-month fall rate in PD $\approx 46\%$; $^1$ 6-month fall rate in PD $\approx 5 \times$ vs. age-matched adults $^2$

- Postural instability may be poorly responsive to pharmacotherapy $^2,^4$ or DBS $^5$

Adapted tango (AT) improves balance and mobility in individuals with PD

- **Promotes adherence** → counters excessive attrition in PD

- **Adapted for PD** → safety, dynamic balance, internal + external movement cues

- **Effective** → gains on Berg Balance Scale, gait speed, and other clinical measures\(^1\)-\(^4\)
  - *retained for 1 month*\(^1\)

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\(^4\)Duncan RP, and Earhart GM. *Neurorehabil Neural Repair* 26: 132-143, 2012
Neurorehabilitation
How does adapted tango improve postural instability in individuals with PD?
We use a reactive balance paradigm and to identify neural mechanisms of balance.
The sensorimotor response model (SRM) describes mapping from motion to EMG.

**Sensory estimates of center of mass (CoM) motion**

**Sensorimotor Response Model (SRM)**

**Motor responses measured with electromyogram (EMG)**

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Agonists are recruited in proportion to CoM motion

Sensorimotor Response Model (SRM)

Support surface translation

Sensory estimates of center of mass (CoM) motion

Motor responses measured with electromyogram (EMG)
Agonists are recruited in proportion to CoM motion

Sensory estimates of center of mass (CoM) motion

Sensorimotor Response Model (SRM)

Motor responses measured with electromyogram (EMG)
Agonists are recruited in proportion to CoM motion.
Antagonists are also recruited in proportion to CoM motion at the end of the perturbation.

CoM motion at the end of the perturbation is processed by feedback gains, time delay, and delayed scaled CoM, which are then summed to a threshold. This process involves feedback gains $k_d$, $k_v$, and $k_a$ for displacement $d$, velocity $v$, and acceleration $a$, respectively, and a delay function $\delta(t-\lambda)$. The support surface translation activates muscle groups such as the medial gastrocnemius (MG) in response to CoM motion.
Antagonists can also coactivate with agonists at the start of the perturbation

CoM \( d(t) \)

**forward CoM motion**

brainstem & spinal cord

feedback gains \( \delta(t-\lambda) \)

time delay

delayed scaled CoM

sum threshold

\begin{align*}
&d \\
&v \\
a
\end{align*}

\begin{align*}
&k_d \\
&k_v \\
&k_a
\end{align*}

**backward CoM motion**

brainstem & spinal cord

feedback gains \( \delta(t-\lambda) \)

time delay

delayed scaled CoM

sum threshold

\begin{align*}
&d \\
&v \\
a
\end{align*}

\begin{align*}
&k_d \\
&k_v \\
&k_a
\end{align*}

The basal ganglia may selectively inhibit alternate CoM “motor programs”

Brainstem & spinal cord

Forward CoM motion

Brainstem & spinal cord

Delayed scaled CoM

Support surface translation

Medial gastrocnemius (MG)

Impaired motor program selection may result in inappropriate coactivation in PD

If AT improves basal ganglia program selection, we predict:

Clinical mobility measures

pre-AT       post-AT
If AT improves basal ganglia program selection, we predict:

Clinical mobility measures
- Appropriate SRM feedback gains
- Inappropriate SRM feedback gains

pre-AT post-AT
We tracked 9 individuals with PD before and after 3-week AT

- Adapted argentine tango lessons\(^1-^3\)
- 15 lessons / 3 weeks
- 1.5 h / lesson
- ON meds assessment

3-week AT improved clinical scales of balance and mobility in individuals with PD

<table>
<thead>
<tr>
<th>Scale</th>
<th>Maximum Possible Score</th>
<th>Pre (N=9)</th>
<th>Post (N=9)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Unified Parkinson’s Disease Rating Scale</td>
<td>56</td>
<td>30±5</td>
<td>28±4</td>
<td>0.189</td>
</tr>
<tr>
<td>Motor Subscale III</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Berg Balance Scale</td>
<td>56</td>
<td>50±7</td>
<td>54±4</td>
<td>0.028*</td>
</tr>
<tr>
<td>Fullerton Advanced Balance Scale</td>
<td>40</td>
<td>27±8</td>
<td>31±6</td>
<td>0.004*</td>
</tr>
<tr>
<td>Dynamic Gait Index</td>
<td>24</td>
<td>19±4</td>
<td>21±3</td>
<td>0.09</td>
</tr>
</tbody>
</table>

3-week AT reduced antagonist SRM gains and increased agonist SRM gains

Subject 3
H&Y 2.5

CoM
\(d(t)\)

TA
MG

support surface translation

Pre-AT

\(k_d, k_i, k_a\)

Kp=0.024, Kv=0.054, Ka=0.661, Td=0.100
Kp=0.000, Kv=0.000, Ka=0.000, Td=0.100
bias=0.093, R²=0.553, VAF=0.759

Kp=0.035, Kv=0.036, Ka=0.507, Td=0.100
Kp=0.000, Kv=0.000, Ka=0.000, Td=0.100
bias=0.369, R²=0.559, VAF=0.925

---

EMG (mean)
EMG (trials)
SRM fit
Appropriate SRM Component
Inappropriate SRM Component

---

0.0
1.0

5 cm

0.5 Hz
3-week AT reduced antagonist SRM gains and increased agonist SRM gains

Subject 3
H&Y 2.5

CoM
$d(t)$

TA

MG

support surface translation

Pre-AT

Post-AT
3-week AT reduced antagonist SRM gains and increased agonist SRM gains

Subject 3
H&Y 2.5

CoM
$d(t)$

TA

MG

support surface translation

Pre-AT

Post-AT

Young Healthy

$K_p=0.024$, $K_v=0.054$, $K_a=0.661$, $T_d=0.100$
$K_p=0.000$, $K_v=0.000$, $K_a=0.000$, $T_d=0.100$
$\text{bias}=0.093$, $R^2=0.553$, $VAF=0.759$

$K_p=0.000$, $K_v=0.000$, $K_a=0.000$, $T_d=0.100$
$K_p=0.035$, $K_v=0.036$, $K_a=0.507$, $T_d=0.100$
$\text{bias}=0.369$, $R^2=0.559$, $VAF=0.925$

$K_p=0.020$, $K_v=0.036$, $K_a=1.331$, $T_d=0.100$
$K_p=0.036$, $K_v=0.012$, $K_a=0.001$, $T_d=0.100$
$\text{bias}=0.045$, $R^2=0.625$, $VAF=0.825$

$K_p=0.014$, $K_v=0.032$, $K_a=4.415$, $T_d=0.099$
$K_p=0.012$, $K_v=0.002$, $K_a=0.001$, $T_d=0.095$
$\text{bias}=0.185$, $R^2=0.719$, $VAF=0.905$

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3-week AT reduced antagonist SRM gains and increased agonist SRM gains

**Subject 3**
**H&Y 2.5**

---

**Pre-AT**

- **TA**
  - \( k_d k_v k_a \)
  - \( K_p=0.024, K_v=0.054, K_a=0.661, T_d=0.100 \)
  - \( K_p=0.000, K_v=0.000, K_a=0.000, T_d=0.100 \)
  - bias=0.093, \( R^2=0.553 \), \( VAF=0.759 \)

- **MG**
  - \( k_d k_v k_a \)
  - \( K_p=0.000, K_v=0.000, K_a=0.000, T_d=0.100 \)
  - \( K_p=0.035, K_v=0.036, K_a=0.507, T_d=0.100 \)
  - bias=0.369, \( R^2=0.559 \), \( VAF=0.925 \)

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**Post-AT**

- **TA**
  - \( k_d k_v k_a \)
  - \( K_p=0.020, K_v=0.036, K_a=1.331, T_d=0.100 \)
  - \( K_p=0.036, K_v=0.012, K_a=0.001, T_d=0.100 \)
  - bias=0.045, \( R^2=0.625 \), \( VAF=0.825 \)

- **MG**
  - \( k_d k_v k_a \)
  - \( K_p=0.014, K_v=0.032, K_a=4.415, T_d=0.099 \)
  - \( K_p=0.012, K_v=0.002, K_a=0.001, T_d=0.095 \)
  - bias=0.185, \( R^2=0.719 \), \( VAF=0.905 \)
3-week AT reduced antagonist SRM gains and increased agonist SRM gains

Subject 1
H&Y 2

CoM
$d(t)$

TA

MG

support surface translation

Pre-AT

Post-AT

$K_p=0.001, K_v=0.057, K_a=2.433, T_d=0.100$

$K_p=0.001, K_v=0.057, K_a=2.433, T_d=0.100$

$K_p=0.000, K_v=0.000, K_a=0.000, T_d=0.100$

bias=0.194, R$^2=0.610$, VAF=0.834

$K_p=0.000, K_v=0.000, K_a=0.000, T_d=0.100$

bias=0.021, R$^2=0.729$, VAF=0.826

$K_p=0.000, K_v=0.000, K_a=0.000, T_d=0.100$

bias=0.258, R$^2=0.530$, VAF=0.849

$K_p=0.001, K_v=0.054, K_a=1.092, T_d=0.100$

bias=0.228, R$^2=0.138$, VAF=0.840

$K_p=0.001, K_v=0.000, K_a=0.428, T_d=0.100$

bias=0.228, R$^2=0.138$, VAF=0.840

UPDRS H&Y BBS FAB DGI
**Deficit**

Impaired program selection by basal ganglia may lead to postural instability in PD

**Neurorehabilitation**

Adapted tango may improve postural instability by improving program selection
Acknowledgments

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