The VTNE may have several questions on receptors, drugs, and the effects of different transmitters on the body; this topic can be very confusing. The goal of this PowerPage is to simplify this information for you and to make it easier to understand.

The body has different receptors in various locations throughout the body that are activated by certain chemicals or transmitters. There are 2 parts of the autonomic nervous system that need to be understood in order to understand the effects of these transmitters on the receptors.

**Sympathetic nervous system**

This is the “fight or flight” system. To best remember these effects, think about what happens when you are frightened.

- Increased heart rate
- Increased blood pressure
- Constriction of peripheral vessels (blood flow shifts to vital organs)
- Decreased blood flow to the GI tract (you don’t need to digest while you are in flight mode)
- Dilation of the bronchioles in the lungs (to help with increased oxygen flow to tissues)
- Dilated pupils (think about how dilated a scared cat’s pupils become in the clinic)

Activation on these adrenergic receptors is via epinephrine (adrenaline) and norepinephrine. Since the receptors for the sympathetic nervous system are termed ‘adrenergic’ (think “adrenaline”), an agonist drug (meaning, ‘having the same affect’) would bind to these receptors and cause the effects listed above.

**Parasympathetic nervous system**

This is the “rest and digest” system. To best remember these, think about what happens when you are relaxed. This is essentially opposite of what the sympathetic nervous system does; therefore, activation on these receptors antagonizes the effects of the sympathetic nervous system.

- Slower heart rate
- Increased blood flow to the GI tract (for digestion)
- Decreased bronchiole diameter
- Very little action on peripheral blood vessels and thus doesn’t change the vasoconstriction level
- Normal pupil size

Activation on these cholinergic receptors is via acetylcholine. There are two different types of cholinergic receptors, called muscarinic (all parasympathetic and giving the signs listed above) and nicotinic (found in both the sympathetic and parasympathetic systems, which are competing for activation and can cause a mix of the signs listed from both categories).
Autonomic vs. Somatic

These 2 parts of the nervous system (sympathetic and parasympathetic) we have no control over; this is why it is termed the **autonomic nervous system** (think automatic: just happens on its own).

There is one other branch called the **somatic nervous system**: this is our **voluntary** system. This system is associated with the **nicotinic** receptors as discussed above. This mostly involves activation upon **skeletal muscles** (think about us moving our arms and legs, etc. voluntarily).

Types of Receptors

When stimulated, adrenergic receptors cause effects associated with the sympathetic nervous system as discussed above. An **agonist drug produces the same effect; an antagonist drug produces the opposite effect**. Sometimes a drug can have a “partial” effect, being called a ‘partial agonist’. The term ‘sympathomimetic’ means it is mimicking the **sympathetic** nervous system.

<table>
<thead>
<tr>
<th>Receptor:</th>
<th>Effect of stimulation:</th>
<th>Common drug <strong>agonists</strong>:</th>
</tr>
</thead>
<tbody>
<tr>
<td>alpha-1</td>
<td>Causes constriction of blood vessels; increases blood pressure</td>
<td>epinephrine, methoxamine</td>
</tr>
<tr>
<td>alpha-2</td>
<td>Helps regulate norepinephrine release</td>
<td>dexmedetomidine, xylazine, romifidine</td>
</tr>
<tr>
<td>beta-1</td>
<td>Located in the heart: causes increased heart rate and contraction</td>
<td>dobutamine</td>
</tr>
<tr>
<td>beta-2</td>
<td>Relaxation of smooth muscle in cardiac blood vessels, skeletal muscle, arterioles, and bronchioles in the lungs</td>
<td>albuterol, terbutaline (mostly used in treating asthma)</td>
</tr>
</tbody>
</table>

In this chart you can see that the listed drugs are **agonists** of those receptors. Therefore, consider the reversal agents for some of these drugs. For example, **the reversal for xylazine is yohimbine**. This means that yohimbine is an alpha-2 **antagonist** (because it is reversing or causing the opposite effects of xylazine, which is an alpha-2 agonist).

If a patient had high blood pressure and rapid heart rate, what could be given to counteract this condition? The answer is a **beta-1 antagonist** (or beta-blocker); a **beta-1 antagonist would relax the heart** (or have the opposite effect than that of dobutamine, which is a beta-1 agonist). Examples of beta-blocker medications are atenolol, sotalol, and propranolol. Some are selective for beta-1, and some have effects on both types of beta-receptors.

Opioids and mu receptors

There is another type of receptor to be aware of. In veterinary medicine, analgesic drugs are frequently used. Opioid receptors are found in the central nervous system, GI, urinary tract, and in smooth muscles. The main receptors for opioids are mu, kappa, and delta; we are going to focus on the mu (µ) receptors. **Remember that morphine-type drugs are mu-agonists.**

<table>
<thead>
<tr>
<th>Classification of mu stimulation:</th>
<th>Opioid Drug Example:</th>
</tr>
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<tbody>
<tr>
<td><strong>agonists</strong></td>
<td>morphine, hydromorphone, meperidine</td>
</tr>
<tr>
<td><strong>partial agonists</strong></td>
<td>butorphanol, buprenorphine</td>
</tr>
<tr>
<td><strong>antagonists</strong> (think reversal agents)</td>
<td>naloxone</td>
</tr>
</tbody>
</table>
References