

## Conferences to Attend in 2017

The year 2017 brings access to knowledge and research collaboration through a series of conference on health care and pharmacy at different locations throughout the world. Some of the upcoming conferences are listed below, visit website for further information. Attend and participate those that best matches your interest and field of research.

**10TH INTERNATIONAL CONFERENCE AND EXHIBITION ON PHARMACEUTICS & NOVEL DRUG DELIVERY SYSTEMS**

**March 13-15, 2017. London, UK**

**10TH ASIA-PACIFIC PHARMA CONGRESS**

**May 08-10, 2017 Singapore**

**INTERNATIONAL CONFERENCE AND EXHIBITION ON NANOMEDICINE AND DRUG DELIVERY**

**May 29-31, 2017 Osaka, Japan**

**4TH ANNUAL CONGRESS ON DRUG DISCOVERY & DESIGNING**

**July 03-05, 2017 Bangkok, Thailand**

**8TH GLOBAL PHARMACOVIGILANCE & DRUG SAFETY SUMMIT**

**July 10-11, 2017 Jakarta, Indonesia**

**8TH WORLD CONGRESS ON PHARMACOLOGY AND TOXICOLOGY**

**July 24-26, 2017 Melbourne, Australia**

**4TH INTERNATIONAL CONFERENCE ON CLINICAL TRIALS**

**September 11-13, 2017 San Antonio, USA**

**INTERNATIONAL CONFERENCE ON BIOTECH PHARMACEUTICALS**

**October 23-25, 2017 Paris, France**

Source: <http://www.pharmaceuticalconferences.com>

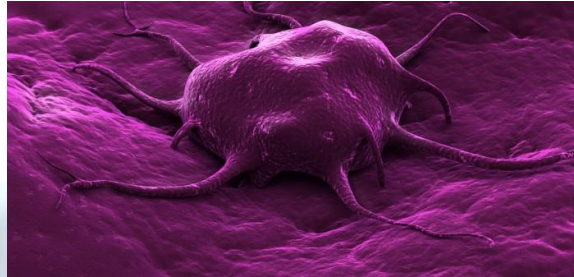
## TSRI Scientists Devise New Approaches to Personalized Medicines

Scientists in the Florida campus of The Scripps Research Institute (TSRI) have developed broad methods to design precision medicines against currently incurable diseases caused by RNA. RNA carries out thousands of essential functions in cells, but many RNAs cannot act in controlled ways and thus cause disease. For decades, scientists have tried to develop drug candidates that target human RNAs, but they have been hampered by an inability to achieve sufficient selectivity (to reduce the potential of side effects) and potency (ensuring effectiveness). According to researchers, “We present for the first time multiple solutions to this long-standing problem. With the precision of a surgeon’s scalpel, we have shown that small molecules can be designed to seek out and destroy only disease-causing RNAs. Further, we developed novel chemical approaches to use a disease-causing RNA to help make its own drug by using that RNA as a catalyst for drug synthesis at the needed site. It is like having your physician place a drug at the right place without exposing healthy cells.” Although these studies have broad implications for RNA diseases in general, they were demonstrated on myotonic dystrophy type 1, an incurable inherited disorder that involves progressive muscle wasting and weakness. It is caused by an RNA defect known as a “triplet repeat,” a series of three nucleotides repeated more times than normal in an individual’s genetic code, in this case, a cytosine-uracil-guanine (CUG) triplet. In many genetic diseases, there are two copies of the problem gene—a mutant copy that causes a disease and a normal copy that a cell needs to survive. Selective recognition of the diseased gene product has not been possible before. This new study demonstrates that designer small molecules can selectively recognize larger, disease-associated repeats (alleles) over shorter, normal ones. “All approaches show precise recognition of toxic r(CUG) repeats and, more importantly, they showed that the mutant repeat is the *sole* target.” The work also offers an innovative way to track the movement of RNA in a diseased cell via imaging. “We probed disease-causing RNA using a technique called fluorescence lifetime imaging—a sensitive technique to measure fluorophore binding,” said Max Planck’s Ryohei Yasuda. “We were very excited when we observed a huge difference in signal from their probes between disease cells and normal cells under our microscope technique.” Max Planck’s Lesley Colgan added,

Source: <https://www.labroots.com/trending/health-and-medicine/4776/tsri-scientists-devise-approaches-personalized-medicine>

## Study Unmasks New Gene Culprit in Deadly Brain Cancer

**A** new study of glioblastoma implicates a gene known as NAMPT which is part of a pathway involved in energy production and cellular aging, helps the brain cancer to survive and proliferate, even in the face of radiation. Glioblastoma multiforme is notorious for being inoperable, and for evading other means of treatment like radiation and chemotherapy, over 70 percent of patients diagnosed with this type of deadly brain cancer rarely survive past the two-year mark. In studying human glioblastoma cells, researchers from the Washington University noticed that the aggressive tumor cells appeared to have overexpression of the NAMPT gene. Tumor cells are fueling their growth from the energy production cycles in the body. "If you target the NAD<sup>+</sup> pathway, you can disrupt the ability of the cancer stem cells to self-renew, and you can also make them more sensitive to radiation treatment," said Albert Kim, assistant professor at Washington University. "In a patient, that could mean that if you suppress the pathway, the same dose of radiation may be more effective at separate experiment, Kim and his team confirmed that the opposite expression of NAMPT were likely to be stunted in growth. Furthermore, inhibiting NAMPT also made the cells more susceptible to cell death via radiation. Interestingly, NAMPT and the NAD<sup>+</sup> pathway has been of interest to researchers who study cellular aging. Specifically, a byproduct molecule of NAMPT, known as nicotinamide mononucleotide (NMN), appears to slow aging in mice. "There's a lot of buzz about taking NAD<sup>+</sup> precursors for their anti-aging effects, which is based on a lot of great science," said Kim. "I don't know if taking NAD<sup>+</sup> precursors makes existing tumors grow faster, but one implication of our work is that we don't yet fully understand all of the consequences of enhancing NAD<sup>+</sup> levels." Cancer cells are often thought of as being immortal as they seem to have bypassed the internal mechanism for normal cell death. This poses an interesting link between glioblastoma multiforme, NAMPT, and aging.



Source: <https://www.labroots.com>

## Long-Term Acetaminophen and NSAID Use Tied to Hearing Loss

**R**egular, long-term use of acetaminophen and nonsteroidal anti-inflammatory drugs (NSAID) is associated with a modestly elevated risk for hearing loss in women, suggests an American Journal of Epidemiology study. Over 55,000 women aged 44–69 in the Nurses' Health Study answered questions about incident hearing loss and how often they took aspirin, acetaminophen, and NSAIDs. During 873,000 person-years' follow-up, nearly 19,000 women said they developed hearing loss. After multivariable adjustment, regular NSAID and acetaminophen use (2 or more days per week) for more than 6 years was associated with incident hearing loss, compared with less than 1 year of use (relative risks, 1.10 and 1.09). Aspirin use showed no association. The authors conclude: "Considering the high prevalence of analgesic use and the high probability of frequent and/or prolonged exposure in women of more advanced age, our findings suggest that NSAID use and acetaminophen use may be modifiable risk factors for hearing loss."

Source:

<http://www.jwatch.org/>

## CDC Publishes Recommendations for 2-Dose Schedule of HPV Vaccine

**T**he CDC has published its human papillomavirus in *MMWR*. The CDC first announced the recommendation in October. For children who start the 14, two doses of the nine-valent vaccination is recommended at ages 6 to 12 months after the efficacy equivalent to that of three for females and 15 through 21 for patients should still receive three 1 to 2 months after the first, and the

Source: <http://www.jwatch.org/>



recommendations on using two doses of (HPV) vaccine — instead of three — announced the recommendation in HPV vaccine series between ages 9 and vaccine are recommended. Routine 11 to 12 years. The second dose should first. In this age group, two doses have an older patients (aged 15 through 26 males) and immunocompromised doses. The second dose should be given third dose given 6 months after the first.