Health Matters

# India's growing role in fentanyl trade: Concern for global health

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A recent intelligence report from the United States highlights India's increasingly prominent role in the global illegal fentanyl trade, a shift that is expected to raise significant concerns in New Delhi. This new development coincides with heightened pressure from the US administration, under President Donald Trump, which has used tariffs as leverage against countries it accuses of insufficient action in combating the flow of fentanyl into the United States.

Fentanyl, a potent synthetic opioid, has been the leading cause of overdose deaths in the US, fuelling an ongoing opioid crisis that remains a high-priority issue for policymakers. According to CNN, fentanyl has been the primary drug involved in overdose fatalities in the US in recent years. This epidemic has significantly impacted public health, leading to a surge in both drug-related deaths and emergency medical responses.

While China has historically been the dominant source of both legal fentanyl supplies and illicit precursor chemicals, recent shifts have brought India into the spotlight. The 2025 Annual Threat Assessment (ATA) report, published by the Office of the Director of National Intelligence (ODNI), indicates that India's role in the illegal fentanyl trade is rapidly increasing. The report states, "Nonstate groups are often enabled, both directly and indirectly, by state actors, such as China and India, as sources of precursors and equipment for drug traffickers." While China remains the primary supplier of illicit fentanyl precursors and pill-pressing machinery, India is now identified as a significant secondary source.

This shift is concerning, given India's well-established position as a global leader in pharmaceutical manufacturing, notably in the production of generic drugs and vaccines. The country's pharmaceutical industry, which supplies a substantial portion of the world's medicines, now faces scrutiny due to its role in the supply chain for fentanyl precursors. According to last year's ATA report, Mexico's drug cartels have increasingly sourced precursor chemicals from India, albeit to a lesser extent than from China.

Fentanyl, originally developed as a potent analgesic for severe pain management, has a highly adaptable chemical structure that allows for the creation of numerous analogs – many of which are far more potent than the original drug. Since its introduction into the illicit drug market in 1979, fentanyl analogs have been used as adulterants in street drugs, often leading to fatal overdoses. The illicit production and distribution of fentanyl have expanded dramatically in recent years, with overdose deaths in the US increasing fourfold, from 8,050 in 1999 to 33,091 in 2015.

The scale of fentanyl-related fatalities has continued to rise, culminating in over 100,000 drug overdose deaths from May 2020 to April 2021, with synthetic opioids, including fentanyl and its analogs, accounting for more than 64% of these deaths. Illegally manufactured fentanyl is synthesized in countries such as China, Mexico, and increasingly, India, before being smuggled into the US in both powder and pill form. China continues to export large quantities of precursor chemicals used to synthesize fentanyl, which are often processed

in clandestine laboratories before being trafficked across the US-Mexico border. The rise in India's involvement in the fentanyl trade presents a significant challenge for global health and international law enforcement. As fentanyl-related overdoses continue to claim lives, the U.S. and other countries must work collaboratively with India to address the underlying supply chains fueling this deadly epidemic. Enhanced cooperation, stronger regulatory frameworks, and a more robust commitment to tackling drug trafficking are critical in mitigating the impact of fentanyl and its analogues on global public health.

# Time-restricted eating affects weight loss

Time-restricted eating is the latest craze for people looking to lose weight, but whether it works is still the calorie-burning question.

A new study from the University of Mississippi shows that when healthy adults pair an eight-hour eating window with regular exercise, they lose more fat - without sacrificing lean muscle - compared to exercise alone, according to a study published recently.

"We saw that this did lead to more fat loss and reduced body fat percentage over time when healthy adults were following both exercise with time-restricting eating compared to those who were only exercising for at least 4 weeks," said Nadeeja Wijayatunga, assistant professor of nutrition and hospitality management.

"It is important to note lean mass preservation."

Wijayatunga and Michael Hays, tactical dietitian and recent Ole Miss graduate, began their study after seeing the dramatic rise in time-restricted eating. Together, they conducted a systematic review and meta-analysis where they analyzed data from 15 studies conducted on time-restricted eating with exercise from the last decade.

Time-restricted eating is one of many diets included in the umbrella term intermittent fasting.

"People like time-restricted eating because they feel it's easier to adhere to because they don't have to think too much," she said. "It's all about time, not calorie-counting or watching out for certain foods."

Intermittent fasting has quickly become one of the most popular diets in the United States, with 12% of Americans having tried it, according to the 2023 International Food and Health Survey.

The science surrounding time-restricted eating, however, is still developing, Hays said.

"For some people, this may be a good technique to help with body composition goals," Hays said. "It's just another tool, but more studies need to come out to really understand how this works in humans."

While the difference between those who exercised while following a time-restricted diet and those who used exercise alone was slight, the two groups in the studies were already very healthy individuals, Hays said.

"In most cases, these were healthy adults," Hays said. "They were already physically fit and already had exercise routines. When you already have athletic, lean people and you decrease their body fat percentage, that's significant."

Time-restricted eating has been criticised for possibly leading to a loss of lean mass - all the muscles, organs and other tissues that make up the human body.

"We need healthy muscles," Wijayatunga said. "Muscles are really important for the body and for your metabolism. If we lose muscle, it may impact our metabolic systems, and it just decreases mobility overall."

In their study, Hays and Wijayatunga found that when paired with exercise, time-restricted eating did not lead to a reduction in lean mass, even for those who lost body fat.

"That's why you want to couple diet with exercise," Hays said. "When you're losing weight, you never want to lose lean tissue. You want to lose fat."

While the results are promising, Wijayatunga warned that much research is needed to confirm the findings. While this study focused on healthy, active subjects, there is a need to understand how this would impact people who are not in shape and exercising regularly.

# Scientists confirm link between dopamine & cognition

For the first time, scientists have confirmed a neurobiochemical link between dopamine and cognitive flexibility, according to new research published recently. PET imaging shows that the brain increases dopamine production when completing cognitively demanding tasks, and that the more dopamine released, the more efficiently the tasks are completed. Armed with this information, physicians may soon be able to develop more precise treatment strategies for neurological and psychiatric disorders.

Cognitive flexibility is the ability to adapt one's thinking and behavior appropriately to a changing environment and is considered an aspect of executive function.

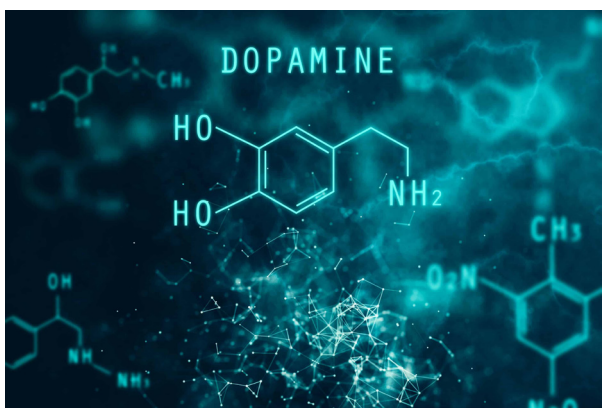
Cognitive flexibility differs among people and is reported to be impaired in several psychiatric and neurologic disorders, such as depression, post-traumatic stress disorder, addiction, anxiety disorder, schizophrenia, Parkinson's disease, and attention-deficit/hyperactivity disorder.

"At the neurotransmitter level, the dopamine system has been linked to cognitive flexibility. A direct neurochemical response to cognitive flexibility, however, has yet to be shown," said Isabelle Miederer, PhD, associate professor in experimental nuclear medicine in the department of nuclear medicine at University Medical Center Mainz, Germany.

"In our study, we sought to examine the release of dopamine in real-time by performing PET scans while individuals completed behavioral flexibility tasks."

Eighteen participants were scanned with the D2/3 receptor ligand 18F-fallypride in a two-part block study design.

In the first part, participants performed two tasks consecutively on a computer screen without




rule switching while undergoing PET imaging.

In the second part of the PET scan, participants had to switch flexibly between two task rules.Dopamine release was calculated using the linearised simplified reference region model which compares the two task blocks with each other.

PET imaging analysis showed a displacement of 18F-fallypride in the ventro-medial prefrontal cortex during the task switching (higher cognitive demand) part of the study, which is assumed to be the release of dopamine.

Results also showed that the greater dopamine release, the more efficient participants were in switching between tasks."The present findings emphasise the significance of dopamine in cognitive flexibility," said Mathias Schreckenberger, MD, head of the department of nuclear medicine at University Medical Center Mainz.

"They are consistent with the results of previous clinical studies indicating that dopamine deficiency in disorders such as Parkinson's disease may cause behavioral deficits in cognitive flexibility.""Looking forward, it is expected that the results of the study will contribute to a better understanding of the neurochemical mechanisms underlying cognitive flexibility and thus facilitate the development of treatment strategies to improve flexibility in neurological and psychiatric disorders," he added.

# Early signs of heart problems linked to smaller brain volumes

People who have early signs of heart problems may also have changes in brain health that can be early signs of dementia, such as loss of brain volume, according to a meta-analysis published recently. The meta-analysis does not prove that early heart problems cause loss of brain cells; it only shows an association.

"This review shows that better heart health is associated with larger brain volumes, suggesting that the preservation of heart function could help maintain brain health and memory and thinking skills during the aging process," said meta-analysis author Frank J. Wolters, MD, PhD, of Erasmus University Medical Center in Rotterdam, the Netherlands.

"These results add to the importance of early detection and treatment of heart problems."

The meta-analysis included seven studies from Europe and the United States with a total of 10,889 participants with an average age of 67. The studies measured early signs of heart problems, including systolic and diastolic dysfunction.

Systolic dysfunction is when the left ventricle of the heart can't contract normally and pump blood efficiently.Diastolic dysfunction is when the left ventricle does not relax properly between heartbeats and fill with blood. The studies also used MRI brain scans to measure brain volumes.

The meta-analysis found that people with moderate to severe systolic dysfunction were more likely to have a smaller total brain volume than people with normal systolic function.

People with diastolic function problems also had a smaller total brain volume and smaller volume in the hippocampus area of the brain, which plays a role in memory.

"The meta-analysis shows that even mild diastolic dysfunction is associated with adverse brain health," Wolters said."Evaluating people who have heart problems - especially impaired diastolic function - for problems with memory and thinking skills could help us detect any cognitive decline early and start interventions."

Wolters noted that additional studies are needed to investigate the relationship between heart health and brain health, particularly to link imaging findings to important health outcomes.A limitation of the meta-analysis was that the majority of participants were white people, so the results cannot be generalised to more diverse populations.

# Respiratory virus causes Kawasaki disease: Study

Research from Stanley Manne Children's Research Institute at Ann & Robert H. Lurie Children's Hospital of Chicago strongly suggests that Kawasaki disease is caused by a single respiratory virus that is yet to be identified. Findings contradict the theory that many different pathogens or toxins could cause this disease that can lead to serious cardiac complications in young children.

"The cause of Kawasaki disease has been a mystery for over 50 years," said Anne Rowley, MD, paediatric infectious diseases expert and scientist at Manne Research Institute at Lurie Children's, who is the lead author of a study published recently. "Our compelling data are a huge step forward and provide a clear direction for the field to identify and sequence the virus that causes Kawasaki disease in susceptible children. This will be critical to advancing the diagnosis, treatment and prevention of Kawasaki disease."

Kawasaki disease is relatively uncommon, affecting mostly children between 6 months and 5 years of age. Lurie Children's sees 50-60 newly diagnosed Kawasaki disease patients a year.

Currently, there is no diagnostic test for Kawasaki disease. Clinical signs include fever, rash, swelling of the hands and feet, irritation and redness of the whites of the eyes, swollen lymph glands in the neck, and irritation and inflammation of the mouth, lips, and throat. Children with Kawasaki disease have a 20 percent chance of developing heart disease, while infants are at higher risk with 50 percent chance of cardiac complications. The standard treatment, intravenous immunoglobulin and aspirin, substantially decreases the risk of heart disease in patients with Kawasaki disease. Steroids may be added for the highest risk patients.

In their study, Dr. Rowley and colleagues prepared antibodies from blood cells of children with Kawasaki disease, in order to see what these antibodies will target in tissue samples of patients who died from the disease. They found that the antibodies recognized so-called inclusion bodies, which are by-products of a virus, in all 20 tissue samples that represented cases from the U.S. and Japan over 50 years.

"We saw the same inclusion bodies targeted in every tissue sample spanning five decades and two continents, which shows that we are dealing with one predominant virus causing Kawasaki disease," said Dr. Rowley. "It appears to be a respiratory virus since the inclusion bodies were in the medium size airways. Going forward, we need to focus on studies of pathology specimens to gain understanding of what is inside the inclusion bodies so that we can identify the Kawasaki disease virus and finally solve the mystery."