



# DAILY NEWS BULLETIN

LEADING HEALTH, POPULATION AND FAMILY WELFARE STORIES OF THE DAY  
Thursday 2020917

## WHO

### **WHO warns of coronavirus momentum as winter looms in north (The Tribune: 2020917)**

<https://www.tribuneindia.com/news/health/who-warns-of-coronavirus-momentum-as-winter-looms-in-north-142115>

Expert advises people at high risk for COVID-19 infections to get flu vaccination

WHO warns of coronavirus momentum as winter looms in north

A "COVID-19 Supply Tent" is seen at Cornell University, New York, Reuters

The World Health Organisation warned on Wednesday that COVID-19, the illness caused by the novel coronavirus, is spreading at a worrying pace in some parts of the northern hemisphere, a few months away from the winter influenza season.

“We are starting to see worrying trends in some countries,” Maria Van Kerkhove, WHO technical lead for COVID-19, said during a social media webcast.

“We are seeing increases in hospitalisations, in intensive care units, particularly in Spain, France, Montenegro, Ukraine and some states of the United States. That is worrying because we have not seen the flu season yet.”

Van Kerkhove also said hospitalisations of people aged 15-49 infected with COVID-19 were increasing in several countries. Dr Mike Ryan, the WHO’s top emergency expert, advised people at high risk for COVID-19 infections to get a flu vaccination.

## Coronavirus, bacteria

### **Coronavirus, bacteria may work together to increase disease severity in some patients (The Tribune: 2020917)**

<https://www.tribuneindia.com/news/health/coronavirus-bacteria-may-work-together-to-increase-disease-severity-in-some-patients-141990>

Coronavirus, bacteria may work together to increase disease severity in some patients  
Photo for representation only.

The severity of COVID-19 in people with obesity and diabetes may be explained by the combined effects of the novel coronavirus and the body's natural community of bacteria -- the microbiota -- working together in the lungs, according to a new study.

The review research, published in the journal eLife, assessed mechanisms linking obesity and diabetes to COVID-19, and suggested that interactions between the novel coronavirus and existing bacterial conditions may explain why people with the co-morbid conditions may often require hospitalisation and ventilation.

"There is rapidly emerging evidence highlighting obesity and type 2 diabetes as key risk factors linked to severity of COVID-19 infections in all ethnic groups, but the detailed underlying connections with these risk factors remain largely unknown," said study co-author Philipp Scherer from the University of Texas Southwestern Medical Center in the US.

"There is a paradox that people with obesity and diabetes are generally known to recover better from lung conditions than others. So, what is it about COVID-19 that makes this group of people more susceptible," Scherer said.

In the research, the scientists revisited the factors and disease pathways that connect obesity and diabetes to the severity of COVID-19 infection.

They found that the mechanisms can be roughly divided into two groups -- those connected with the human cells' ACE2 receptor, and those providing an interaction between COVID-19 and pre-existing bacterial conditions.

The scientists explained that the ACE2 receptor resides on the surface of many cells in the human body, and is involved in regulating blood pressure and the function of blood vessels, and is also used by the virus to enter human cells.

They hypothesised that increased amounts of ACE2 in people with obesity or diabetes makes it easier for the virus to enter cells and increases the viral load -- an important factor in determining disease severity.

Alternatively, the researchers said, an increased shedding of ACE2 in people with obesity causes it to move to the lungs, where the virus could use it.

They believe the body's own microbiota may also be influential in the progression of lung diseases.

According to the scientists, humans carry more than 100 trillion bacteria in the body -- outnumbering the number of our own cells.

They said people with obesity and diabetes are thought to suffer from a body-wide dissemination of bacteria and the substances they produce, which in turn causes low-level continuous inflammation in different tissues.

The scientists are currently assessing how host bacteria might influence COVID-19 severity.

They said one potential culprit could be the lipopolysaccharide (LPS) molecules that bacteria produce, which they said can cooperate with other coronaviruses to induce respiratory distress in pigs.

They noted in the study that the LPS molecules may likely join forces with COVID-19 in humans and trigger a chain of events that causes healthy tissue to transform into scarred tissue.

"While all of these potential mechanisms can contribute to the severity of COVID-19, we believe that one of them plays the predominant role, and that this must be present not only in obese and diabetic patients, but also in other groups of increased risk in COVID-19," Scherer explained.

The scientists said a combined deficiency in ACE2 caused by COVID-19, together with obesity or diabetes, may lead to impaired gut barrier function, allowing bacteria and their toxins to leak into the circulation.

They believe these bacteria and toxins may be working with the virus in the lungs to cause more severe injury than either would do alone.

"Our theory is supported by experiments showing that the combination of bacterial and viral infection can lead to a 'cytokine storm' -- an extreme inflammatory reaction -- which is a hallmark of COVID-19," Scherer said.

"Moreover, the involvement of viral-bacterial interactions can also explain the increased risk of severe COVID-19 seen in older people, those with heart disease and in some ethnic groups," he added. PTI

## **Covid-19: What you need to know today**

## **Covid-19: What you need to know today (Hindustan Times: 2020917)**

<https://epaper.hindustantimes.com/Home/ArticleView>

How seriously does one take Dr Li-Meng Yan? And how seriously does one take the paper Unusual Features of the Sars-CoV2 Genome Suggesting Sophisticated Laboratory Modification Rather Than Natural Evolution and Delineation of its Probable Synthetic Route, published by

her and co-authors, under the aegis of the Rule of Law Society and the Rule of Law Foundation, New York, on September 14? As the title suggests, the paper claims the coronavirus was man-made, in a laboratory.

The paper was uploaded on open-source research repository Zenodo, run by CERN, and was reported by Hindustan Times on Wednesday ([bit.ly/33uFyy4](https://bit.ly/33uFyy4)). It wasn't as widely reported as Dr Yan's comments in Loose Women, a segment of a TV show hosted by a UK TV channel, on which she pretty much said the same thing, albeit without any of the scientific arguments - - unsubstantiated ones -- presented in the paper.

Here's what that paper claimed:

One, ZC45, a bat virus, or a closely related variant or mutant, bears a striking similarity with Sars-CoV2, as shown by genome sequencing, with a 94%-100% similarity of key viral proteins.

The spike protein of Sars-CoV2 is essentially a trimer (essentially three parts) each of which has an S1 and S2 part with a furin cleavage site at the boundary between the two. Other research has already established that the human cellular enzyme furin cleaves, or breaks, the S1 and S2 unit at the cleavage site, and that the S1 unit then attaches to the ACE receptor, another protein found on the surface of most human cells. This binding then facilitates the entry of the viral protein into human cells. The virus' ability to bind with the receptor, and the presence of the cleavage site that responds to a common human enzyme, are the reasons Covid-19 is as infective as it is.

Both the furin cleavage site, and the binding ability of the spike protein with the ACE2 receptor aren't natural, the paper argued.

In their preface to this scientific hypothesis, the authors also claim that the process of creating such a virus in a laboratory could take only six months. They ask for further research and investigation into the origin of the virus. Even if their hypothesis is subsequently proven erroneous, this is a recommendation that no can argue with — the origin of the virus needs to be investigated, not so much to assign blame (although there will be some that too), but to prepare for the next virus and the next pandemic.

Dr Yan, currently in the US, where she fled to in late April, is a virologist who used to work at the University of Hong Kong School of Public Health, and who has for long claimed that China knew of the virus and the fact that human-to-human transmission of the infection was happening, long before it let on. Her claims on the virus being man-made are more recent.

Interestingly, a March paper in Nature titled The Proximal Origin of Sars-Cov2, authored by Kristian G Andersen of California's Scripps Research Institute, argued, again picking on the same two distinctive features of Sars-CoV2, that the virus was natural. The viral protein showed a high "affinity" to bind with the receptor, they said, but this interaction wasn't "ideal" or "optimal". In plain English this meant that if anyone had set out to engineer the virus, they would have picked the "ideal" binding relation, not just another optimal one. The paper also said that there were other coronaviruses that had similar "cleavage sites" and that this wasn't unique to Sars-CoV2.

However, the two papers differ in one significant aspect. The one published in Nature said “the genetic data irrefutably show that Sars-CoV2 is not derived from any previously used virus backbone”. Dr Yan’s said (again, without substantiation that) a “ genomic sequence analysis reveals that ZC45, or a closely related bat coronavirus, should be the backbone used for the creation of Sars-CoV2”.

Dr Yan’s claims are also being seen through a political lens, with scientists in the US pointing out that the two non-profits that published the paper were linked to Steve Bannon, former Trump adviser and former executive chairman of the far-right Breitbart News, casting aspersions on the study’s findings.

Clearly, only further research and investigation can shed light on the origin of the virus which has thus far infected 29,927,685 and killed 942,564 around the world. India ended Wednesday with 5,115,846 cases and 83,230 deaths.

But as Vivek Wadhwa, a columnist for this paper, a top technology thinker, and distinguished fellow at Harvard Law School’s Labor and Worklife Program, said in a recent article in Foreign Policy: “If genetic engineering wasn’t behind this pandemic, it could very well unleash the next one.” That’s because, “genetic engineering — with all its potential for good and bad — has become democratised”, Wadhwa wrote.

“Thanks to a technological revolution in genetic engineering, all the tools needed to create a virus have become so cheap, simple, and readily available that any rogue scientist or college-age biohacker can use them.”

## **Oxford COVID-19 vaccine**

### **Serum Institute gets DCGI nod to resume clinical trial of Oxford COVID-19 vaccine (The Hindu: 2020917)**

<https://www.thehindu.com/sci-tech/health/serum-institute-gets-dcgi-nod-to-resume-clinical-trial-of-oxford-covid-19-vaccine/article32617410.ece>

82,961 COVID-19 patients were cured and discharged in the last 24 hours

The Drugs Controller General of India (DCGI) has now allowed Serum Institute of India (SII) to resume the previously paused clinical trial of the Oxford COVID-19 vaccine in the country, confirmed a senior Health Ministry official.

## **Coronavirus**

### **Coronavirus | dining out raises risk of infection, reports U.S. study (The Hindu: 2020917)**

<https://www.thehindu.com/sci-tech/health/dining-out-raises-risk-of-infection-reports-us-study/article32593014.ece>

Positive adults were twice as likely to report eating out 14 days prior to getting infection. Restaurant and pub exposure were cited as important risk factors associated with SARS-CoV-2 infection, according to an analysis by the U.S. Centres for Disease Control and Prevention (U.S.CDC).

### **Coronavirus (The Times of India: 2020917)**

<https://timesofindia.indiatimes.com/india/coronavirus-live-updates-india-cases-cross-5-million-recoveries-highest/liveblog/78157924.cms>

# TRACKING COVID-19



September 17, 2020 11:00 am (IST)

TOTAL CASES (GLOBAL)

**29.76mn**

ACTIVE

**8.59mn**

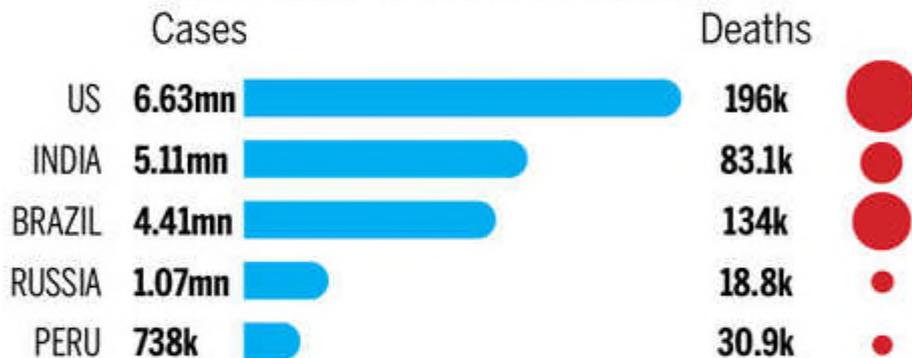
RECOVERIES

**20.22k**

DEATHS

**939k**

## MOST AFFECTED COUNTRIES



Source: Johns Hopkins University

## INDIA UPDATE

- India registers spike of 97,894 new cases & 1,132 deaths in last 24 hours
- One in three Delhiites has Covid antibodies, reveals latest sero survey
- Over 3 lakh Indian workers returned to India under Vande Bharat Mission: Govt
- Union minister Nitin Gadkari tests positive for Covid-19
- Kerala allows migrant workers who are Covid positive but asymptomatic to work in exclusive areas

## **Hypertension**

### **Hypertension most common comorbidity among Covid patients: AIIMS doctor (New Kerala: 2020917)**

<https://www.newkerala.com/news/2020/164923.htm>

The All India Institute of Medical Sciences (AIIMS), Delhi, recorded underlying health conditions or comorbidities in 98.5 per cent of coronavirus patients admitted to the hospital for treatment in September with hypertension being the most common form.

There is a grave interlink between comorbidities and coronavirus infection. Patients with hypertension, diabetes, coronary heart disease, chronic obstructive lung, carcinoma or chronic kidney disease have higher chance of succumbing to the disease, according to public health experts.

According to Animesh Ray, Assistant Professor in the Department of Medicine at AIIMS, "In September, 137 patients were admitted to AIIMS, and comorbidities were present in 98.5 per cent of them. There was presence of comorbidities in almost all the patients, barring two."

Ray said that hypertension was the most common form of ailment in the patients, followed by diabetes and malignancy.

"One in four patients had multiple comorbidities. Out of 137, 128 were discharged while nine succumbed to the disease, out of which six were asymptomatic or had mild infection, while three were severely ill," Ray said during the AIIMS National Combined Grand Rounds on Covid-19.

Hypertension is defined as blood pressure above 140/90, and is considered severe if the pressure is above 180/120. High blood pressure often has no symptoms. Over time, if untreated, it can cause health conditions, such as heart disease and stroke.

AIIMS Director Randeep Guleria, who is also the part of a core team monitoring the pandemic, also said that comorbidities not only lead to a poor prognosis in terms of morbidity and mortality, but sometimes they also get aggravated because of underline Covid-19.

"Diabetes has emerged as an important comorbidity. It can lead to poor prognosis in terms of morbidity and mortality, especially where diabetes is not well-controlled. It has been well said that a collision of two pandemics -- Covid-19 pandemic and diabetes pandemic -- has led to worse outcomes in these patients."

Guleria added that there is a bi-directional flow as far as diabetes and Covid-19 is concerned. Individuals who have diabetes have more severe Covid-19 infection.

"There are some data which suggest that it may aggravate to lead to the development of Type-1 diabetes. Anyone who has diabetes, infection control becomes an issue for him/her."

## **Regenerative medicine**

### **Study focuses on new discovery in regenerative medicine (New Kerala: 2020917)**

<https://www.newkerala.com/news/2020/164906.htm>

An international collaboration involving Monash University and Duke-NUS researchers have made an unexpected world-first stem cell discovery that may lead to new treatments for placenta complications during pregnancy.

While it is widely known that adult skin cells can be reprogrammed into cells similar to human embryonic stem cells that can then be used to develop tissue from human organs -- known as induced pluripotent stem cells (iPSCs) -- the same process could not create placenta tissue.

iPSCs opened up the potential for personalised cell therapies and new opportunities for regenerative medicine, safe drug testing, and toxicity assessments, however little was known about exactly how they were made.

An international team led by ARC Future Fellow Professor Jose Polo from Monash University's Biomedicine Discovery Institute and the Australian Research Medicine Institute, together with Assistant Professor Owen Rackham from Duke-NUS in Singapore, examined the molecular changes the adult skin cells went through to become iPSCs. It was during the study of this process that they discovered a new way to create induced trophoblast stem cells (iTSCs) that can be used to make placenta cells.

This exciting discovery, also involving the expertise of three first authors, Dr Xiaodong Liu, Dr John Ouyang and Dr Fernando Rossello, will enable further research into new treatments for placenta complications and the measurement of drug toxicity to placenta cells, which has implications during pregnancy.

"This is really important because iPSCs cannot give rise to placenta, thus all the advances in disease modelling and cell therapy that iPSCs have brought about did not translate to the placenta," Professor Polo said.

"When I started my PhD five years ago our goal was to understand the nuts and bolts of how iPSCs are made, however along the way we also discovered how to make iTSCs," said Dr Liu.

"This discovery will provide the capacity to model human placenta in vitro and enable a pathway to future cell therapies," commented Dr Ouyang.

"This study demonstrates how by successfully combining both cutting edge experimental and computational tools, basic science leads to unexpected discoveries that can be transformative," Professor Rackham said.

Professors Polo and Rackham said many other groups from Australian and international universities contributed to the study over the years, making it a truly international endeavour.

## **Dementia**

### **PTSD may double risk of dementia: Study (New Kerala: 2020917)**

<https://www.newkerala.com/news/2020/164917.htm>

People who have experienced post-traumatic stress disorder (PTSD) are up to twice as likely to develop dementia later in life, warn researchers.

For the study, published in the British Journal of Psychiatry, the researchers analysed findings from 13 studies conducted on four continents on PTSD and dementia risk.

"Our study provides important new evidence of how traumatic experiences can impact brain health, and how the long-term effects of trauma may impact the brain in many ways increasing vulnerability to cognitive decline and dementia," said study author Vasiliki Orgeta from the University College London (UCL) in the UK.

The research team included data from a total of 1,693,678 people, investigating whether a PTSD diagnosis was associated with increased risk of dementia up to 17 years later.

By pooling data from eight of the studies, the researchers found that people with PTSD faced a 61 per cent higher risk of dementia.

Analysing data from two studies that used different methods, they found that PTSD was associated with double the odds of developing dementia.

The researchers say the risk could be higher than the studies suggest, as PTSD also increases the likelihood of developing other known dementia risk factors, such as depression, social isolation, or elevated alcohol intake.

Most of the studies adjusted for some of these factors, so the overall findings might underestimate the true cost of PTSD.

It remains unclear how PTSD raises dementia risk, but the researchers say it may be related to hypervigilance and recurrent re-experiencing of trauma, contributing to threat and stress-related activity in the brain, while withdrawal from social life may reduce cognitive reserve and resilience.

"Our findings add to a growing body of evidence that dementia can sometimes be prevented by addressing risk factors throughout an individual's life course," said study author Mia Maria Gunak from UCL.

"Here we have identified an additional group of people who face an elevated risk of dementia, who may benefit from further mental health support," Maria added.

## **Mental health**

### **Effective way to increase capacity for mental health (New Kerala: 2020917)**

As anxiety and depression rise during the pandemic, primary care clinics, especially in rural areas, are facing increased patient needs.

A way to address this is to bolster healthcare providers' ability to diagnose and treat patients with common mental health disorders.

Researchers at UW Medicine found that primary-care physicians and rural clinic staff felt more skilled in delivering mental health care if they used a model known as collaborative care.

The results were published on September 14 in the *Annals of Family Medicine* and added to the evidence supporting collaborative care's effectiveness.

In the model, primary-care physicians retain primary responsibility to treat behavioural health disorders with the support of two team members a care manager and a consulting psychiatrist. Consulting psychiatrists provide recommendations on patient care through weekly caseload reviews conducted online.

"We found that primary-care doctors involved in this collaboration got better at diagnosing, prescribing, and working as a team," said lead author Dr Morhaf Al Achkar, associate professor of family medicine at the University of Washington School of Medicine.

In the qualitative study, researchers interviewed 17 clinical, support, and administrative staff at three rural clinics in Washington state. All interviewees said the consultations improved their competence to identify and treat psychiatric disorders.

Researchers concluded that weekly systematic case reviews using telepsychiatry consultation functioned both as a model for patient care and as a workforce training and development strategy.

The study was conducted by Rural PREP (Collaborative for Rural Primary care Research, Education and Practice), a UW School of Medicine-led collaboration funded by the U.S. Health Resources and Services Administration to improve training of rural primary-care professionals.

"The learning was bi-directional," said Al Achkar. Primary-care doctors learned how to better work with patients.

Care managers learned to appreciate how medical issues affect mental health and how to diagnose and assess mental health issues. Consulting psychiatrists learned how to coach a primary-care team.

Al Achkar said clinic staff know the community and share that knowledge to help the consulting psychiatrists. And, he said, clinic staff learned to treat mental health as a chronic disease like hypertension and diabetes.

Collaborative care was developed at the UW School of Medicine. It employs principles of effective chronic illness care Focus on defined patient populations, track these patients in a registry, and measure their progress.

The model was pioneered by the late Dr Wayne Katon, former vice chair of Psychiatry and Behavioral Sciences, who saw a link between depression and physical health. The model has been widely disseminated throughout the United States by the Advancing Integrated Mental Health Solutions (AIMS) Center at the Department of Psychiatry and Behavioral Sciences.

More than 80 clinical trials support the effectiveness of collaborative care in treating behavioural health disorders in primary care and Medicare billing codes and policies in many states support its implementation.

Based on the findings of the recent study, researchers said the additional benefit of increasing the skills of the primary-care workforce should be considered as healthcare organisations consider costs and benefits of implementing collaborative care.

## **Immunological diseases**

### **Study connects hormones to lifetime risk for immunological diseases (New Kerala: 2020917)**

<https://www.newkerala.com/news/2020/164488.htm>

Differences in biological sex can dictate lifelong disease patterns, says a new study by Michigan State University researchers that links connections between specific hormones present before and after birth with immune response and lifelong immunological disease development.

Published in the most recent edition of the Proceedings of the National Academy of Sciences, the study answers questions about why females are at increased risk for common diseases that involve or target the immune system like asthma, allergies, migraines and irritable bowel syndrome (IBS).

The findings by Adam Moeser, Emily Mackey and Cynthia Jordan also open the door for new therapies and preventatives.

"This research shows that it's our perinatal hormones, not our adult sex hormones, that have a greater influence on our risk of developing mast cell-associated disorders throughout the lifespan," said Moeser, Matilda R. Wilson Endowed Chair, a professor in the Department of Large Animal Clinical Sciences and the study's principle investigator.

"A better understanding of how perinatal sex hormones shape lifelong mast cell activity could lead to sex-specific preventatives and therapies for mast cell-associated diseases," added Moeser.

Mast cells are white blood cells that play beneficial roles in the body. They orchestrate the first line of defence against infections and toxin exposure and play an important role in wound healing, according to the study, 'Perinatal Androgens Organize Sex Differences in Mast Cells and Attenuate Anaphylaxis Severity into Adulthood.'

However, when mast cells become overreactive, they can initiate chronic inflammatory diseases and in certain cases, death. Moeser's prior research linked psychological stress to a specific mast cell receptor and overreactive immune responses.

Moeser also previously discovered sex differences in mast cells. Female mast cells store and release more inflammatory substances like proteases, histamine and serotonin, compared with males.

Thus, female mast cells are more likely than male mast cells to kick-start aggressive immune responses. While this may offer females the upper hand in surviving infections, it also can put females at higher risk for inflammatory and autoimmune diseases.

"IBS is an example of this. While approximately 25 per cent of the US population is affected by IBS, women are up to four times more likely to develop this disease than men," said Mackey, whose doctoral research is part of this new publication.

Moeser, Mackey and Jordan's latest research explains why these sex-biased disease patterns are observed in both adults and prepubertal children. They found that lower levels of serum histamine and less-severe anaphylactic responses occur in males because of their naturally higher levels of perinatal androgens, which are specific sex hormones present shortly before and after birth.

"Mast cells are created from stem cells in our bone marrow. High levels of perinatal androgens program the mast cell stem cells to house and release lower levels of inflammatory substances, resulting in a significantly reduced severity of anaphylactic responses in male newborns and adults," Moeser said.

"We then confirmed that the androgens played a role by studying males who lack functional androgen receptors," said Jordan, professor of Neuroscience and an expert in the biology of sex differences.

While high perinatal androgen levels are specific to males, the researchers found that while in utero, females exposed to male levels of perinatal androgens develop mast cells that behave more like those of males.

"For these females, exposure to the perinatal androgens reduced their histamine levels and they also exhibited less-severe anaphylactic responses as adults," said Mackey, who is currently a veterinary medical student at North Carolina State University.

In addition to paving the way for improved and potentially novel therapies for sex-biased immunological and other diseases, future research based will help researchers understand how physiological and environmental factors that occur early in life can shape lifetime disease risk, particularly mast cell-mediated disease patterns.

"While biological sex and adult sex hormones are known to have a major influence on immunological diseases between the sexes, we're learning that the hormones that we are exposed to in utero may play a larger role in determining sex differences in mast cell-associated disease risk, both as adults and as children," Moeser said.

## **Covid-19, bacteria**

### **Covid-19, bacteria together may increase severity in some patients (New Kerala: 2020917)**

<https://www.newkerala.com/news/2020/164483.htm>

The combined effects of the body's natural community of bacteria, also called microbiota, working together with Covid-19 in the lungs could explain the severity of the disease in people with obesity and diabetes, say researchers.

The review, published in the journal eLife, offers important mechanistic insights into why people with obesity and diabetes seem to be at increased risk of developing severe acute respiratory syndrome (SARS) after infection with the Covid-19 virus, and more often require hospitalisation and ventilation.

"There is rapidly emerging evidence highlighting obesity and type 2 diabetes as key risk factors linked to severity of Covid-19 infections in all ethnic groups," said study author Philipp Scherer from the University of Texas Southwestern Medical Centre, Dallas in the US.

In their article, the research team revisit the factors and disease pathways that connect obesity and diabetes to the severity of Covid-19 infection.

The mechanisms can be roughly divided into two groups those connected with the ACE2 receptor, and those providing an interaction between Covid-19 and pre-existing bacterial conditions.

ACE2 resides on the surface of many cells in the human body and is involved in regulating fluid volumes, blood pressure and the function of blood vessels. It is also used by Covid-19 to enter human cells.

One theory is that increased amounts of ACE2 in people with obesity or diabetes makes it easier for the virus to enter cells and increases the viral load -- an important factor in determining disease severity.

Alternatively, increased shedding of ACE2 in people with obesity causes it to move to the lungs, where the virus can use it.

Another factor known to be influential in the progression of lung diseases is our body's microbiota. We carry more than 100 trillion bacteria in our body -- outnumbering the number of our own cells.

The team considered how host bacteria might influence Covid-19 severity.

One potential culprit is the lipopolysaccharides (LPS) that bacteria produce, which have been shown to cooperate with other coronaviruses to induce SARS in pigs.

It is possible that these LPS molecules join forces with Covid-19 in humans and trigger a chain of events that causes healthy tissue to transform into scarred tissue -- as Covid-19 does in the lungs.

"While all of these potential mechanisms can contribute to the severity of Covid-19, we believe that one of them plays the predominant role, and that this must be present not only in obese and diabetic patients, but also in other groups of increased risk in Covid-19," Scherer explained.

## Coronavirus

**8 महीने, 6 करोड़ कोरोना टेस्ट... जानें कैसे कोविड-19 से जंग जीतने की ओर बढ़ रहे भारत के कदम (Hindustan: 2020917)**

<https://www.livehindustan.com/national/story-coronavirus-cases-india-tested-60-million-samples-for-covid-19-in-8-months-3496431.html>

भारत में भले ही कोरोना वायरस संक्रमण के मामले हर दिन बढ़ रहे हैं, मगर यह भी हकीकत है कि इसके जांच की संख्या में भी पहले की तुलना में बड़ा इजाफा हुआ है। भारत में 60 मिलियन यानी 6 करोड़ से अधिक कोरोना वायरस के सैंपल की जांच की जा चुकी है। बुधवार को 11,36,613 कोरोना सैंपल की जांच की गई। इस तरह से देखा जाए तो 6,05,65,728 कोरोना टेस्ट करने में देश को करीब आठ महीने का समय लगा। भारत में पहले कोविड-19 के सैंपल का टेस्ट 23 जनवरी को पुणे स्थित भारतीय आयुर्विज्ञान अनुसंधान परिषद के नेशनल इंस्टीट्यूट ऑफ वायरोलॉजी में किया गया था।

यहां ध्यान देने वाली बात यह है कि जनवरी में भारत के पास सिर्फ एक एनआईवी (नेशनल इंस्टीट्यूट ऑफ वायरोलॉजी) लैब थी। तब से देश ने सरकारी और निजी क्षेत्र में कुल 1,751 लैब को अपने नेटवर्क में जोड़ा है। इनमें से सरकारी 1059 प्रयोगशाला हैं और 659 प्राइवेट प्रयोगशालाएं हैं। नाम न जाहिर होने की शर्त पर एक आईसीएमआर अधिकारी ने कहा कि कोरोना टेस्ट के लिए लैब को दैनिक आधार पर उचित अप्रूवल के बाद जोड़ा जा रहा है।

पिछले तीन सप्ताह से औसतन भारत हर दिन अब एक मिलियन यानी करीब दस लाख कोरोना सैंपलों की जांच कर रहा है। इनमें आरटी-पीसीआर और रैपिड एंटीजेन टेस्ट भी शामिल हैं।

आईसीएमआर द्वारा जारी डेटा के मुताबिक, कोरोना के हुए अब तक के कुल जांच में रैपिड एंटीजेन टेस्ट की भागीदारी 40 फीसदी है। देश में कोरोना टेस्ट की संख्या में लगातार बढ़ोतरी हो रही है। 8 अप्रैल तक कोरोना के कुल 10,000 टेस्ट किए गए थे। 3 मई तक यह आंकड़ा बढ़कर एक मिलियन (दस लाख) हो गया, 10 जून तक पांच मिलियन (पचास लाख), 7 जुलाई तक 10 मिलियन (1 करोड़) और बुधवार को कोरोना टेस्ट 60 मिलियन (6 करोड़) का आंकड़ा पार कर लिया।

केंद्रीय स्वास्थ्य और परिवार कल्याण मंत्रालय के लेटेस्ट आंकड़ों के अनुसार, इस सप्ताह देश में किए गए औसत दैनिक कोविड-19 परीक्षणों में 29 जुलाई से 4 अगस्त के बीच 0.5 मिलियन (5,04,266) की वृद्धि देखी गई है। हालांकि, कुछ राज्यों में कोविड 19 का उच्च पॉजिटिविटी रेट चिंता का विषय है, जहां कोरोना टेस्ट बड़े पैमाने पर हो रहा है। देश में कोरोना वायरस का पॉजिटिव रेट 8.4 फीसदी है। मगर महाराष्ट्र में यही आंकड़ा 21.5 फीसदी, आंध्र प्रदेश में 12.3 फीसदी और कर्नाटक में 12.1 फीसदी है। हालांकि, इन राज्यों में टेस्ट की रफ्तार भी बढ़ी है।

आज यानी गुरुवार को जारी किए गए आंकड़ों के मुताबिक, बीते 24 घंटे के बात करें तो देश में 97 हजार से अधिक नए मामले सामने आए हैं। इसके साथ ही कुल मामलों की संख्या 51 लाख के पार कर गई है। मौत के आंकड़ों पर गौर करें तो बीते कुछ समय से रोजाना एक हजार से अधिक मरीज इस महामरी से मर रहे हैं। स्वास्थ्य और परिवार कल्याण मंत्रालय द्वारा जारी आंकड़ों के मुताबिक, बीते 24 घंटे में कोरोना के 97,894 नए पॉजिटिव केस सामने आए हैं। इसके साथ ही 1132 मरीजों की मौत भी हो गई है।