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SymptomsOrigins and historyModern diagnosisTreatment today. In 1900, Swiss psychiatrist Paul Eugen Bleuler coined the term "schizophrenia." Derived from Greek roots, the word contains "schizo" meaning
 "split" and "phrene," which means "mind." Schizophrenia is frequently stigmatized and misunderstood, often far more than other mental health conditions. A 2010 study found that it can be common for those with schizophrenia to be perceived as dangerous, even though other research suggests most people living with this condition are generally
nonviolent. Looking at the condition's origins can be an important starting point in changing the current stigma and public perception of schizophrenia shout spiritual causes and treatments that some may view as unethical or inhumane. This history may be a main contributor to current stigmas around
schizophrenia and those living with the disorder. According to Tracy McDonough PhD, psychology professor and president of the Schizophrenia Oral History Project, a nonprofit dedicated to archiving the life stories of people with schizophrenia Oral History Project, a nonprofit dedicated to archiving the life stories of people with schizophrenia Oral History Project, a nonprofit dedicated to archiving the life stories of people with schizophrenia Oral History Project, a nonprofit dedicated to archiving the life stories of people with schizophrenia Oral History Project, a nonprofit dedicated to archiving the life stories of people with schizophrenia Oral History Project, a nonprofit dedicated to archiving the life stories of people with schizophrenia Oral History Project, a nonprofit dedicated to archiving the life stories of people with schizophrenia Oral History Project, a nonprofit dedicated to archiving the life stories of people with schizophrenia Oral History Project, a nonprofit dedicated to archiving the life stories of people with schizophrenia Oral History Project, a nonprofit dedicated to archiving the life stories of people with schizophrenia Oral History Project, a nonprofit dedicated to archiving the life stories of people with schizophrenia Oral History Project, a nonprofit dedicated to archiving the life stories of people with schizophrenia Oral History Project, and the life stories of people with schizophrenia Oral History Project, and the life stories of people with schizophrenia Oral History Project, and the life stories of people with schizophrenia Oral History Project, and the life schizophrenia Oral History 
schizophrenia."Theories about the possible causes and treatments for mental health conditions resembling schizophrenia date far back to antiquity. Ancient minds frequently perceived the cause of mental illnesses like schizophrenia as a punishment from gods, or possibly as possession from evil spirits and demons. References to "madness"
resembling schizophrenia date to the Old Testament, and even further. A condition similar to schizophrenia is described in "The Book of Hearts," a chapter dedicated to mental health disorders in "The Ebers Papyrus," an ancient Egyptian medical manuscript dating to 1550 BC. Similar to the Egyptians, ancient Greek "father of medicine" Hippocrates
believed conditions like schizophrenia were rooted in biology, rather than spiritual or metaphysical causes. According to Hippocrates, mental health disorders were caused by imbalances in the "four bodily humors," and could be treated with schizophrenia — like psychosis
and hallucinations — were commonly viewed as proof of demonic possession and sin throughout Europe in the Middle Ages. However, some medieval sources indicate that these mental health conditions were thought to be caused by:imbalances in the bodydiet and alcohol intakeoverworking grief and lossInstitutionalizing those with mental disorders
like schizophrenia in asylums or "fools towers" formally began in medieval Europe. Common treatments for mental illness in the skull, either to relieve pressure or to release demons and spirits. Schizophrenia continued to be viewed as "madness" for hundreds
of years. Until the mid-20th century, treatment for schizophrenia was often experimental at best and cruel and inhumane at worst. McDonough told Psych Central, "Early on, people [with schizophrenia] were believed to be untreatable. They often were locked away in asylums. In Europe, people would visit the asylums like they were going to the zoo.
McDonough continued, "At its core, [schizophrenia's] history is one of not seeing human beings as human beings
included:respecting the person under psychiatric care establishing doctor-patient relationships based on trust and confidentiality decreasing stimuli encouraging routine activity and exercise In humanely treating people with mental health conditions like schizophrenia, Pinel emphasized the need for:hygieneexercisekeeping
detailed case histories and records for each personIn 1893, German psychiatrist Emil Kraepelin coined the term "dementia praecox," meaning "premature dementia," to describe schizophrenia as a progressive and irreversible biological condition with potential toxic causes. His contributions to
studying schizophrenia were far more intentionally scientific and naturalistic than many of his predecessors. Swiss psychiatrist Eugene Bleuler coined the famous "four As" of schizophrenia describing negative symptoms of schizophrenia, which were later
changed to the 5 As. Institutionalizing people with schizophrenia was still a common practice well into the 20th century. Common 20th century treatments for schizophrenia included: insulin coma therapy: repeatedly injecting large amounts of insulin to induce daily comas over a period of weeksMetrazol shock: a potentially fatal form of shock therapy
involving injections of Metrazol (pentylenetetrazol) to trigger convulsions and comaelectroconvulsive therapy: stimulating or shocking the brain with electricity to induce seizures surgery: including frontal lobotomy Eugenics also played a dark role in 20th century schizophrenia treatments. At the time, schizophrenia was feared as a largely genetically
inherited condition. Due to persistent stigma and misunderstanding, many people with schizophrenia were sterilized, often without consent. The first antipsychotics, like Chlorpromazine, were developed and released in the 1950s. These medications are
still prescribed today and are considered "typical antipsychotics." The 1990s saw the development of more sophisticated antipsychotics — to treat schizophrenia. When diagnostic tools, like self-reporting forms, are also used alongside
qualitative clinical assessments, such as: Communication Disturbances IndexThe Positive and Negative Symptom Scale (PANSS) BPRS (Brief Psychiatric Rating Scale) The Mini Mental Status Exam ("The Mini") Schizophrenia is a treatable condition. Doctors and therapists now have a number of tools to help people with this disorder find the best
treatment plans possible for them. Many people with schizophrenia are able to manage their symptoms and live well-balanced, fulfilling lives. Antipsychotic medication is often used continuously in schizophrenia treatment plans to manage psychosis. Two classes of antipsychotic medications are prescribed today for schizophrenia: typical and atypical
antipsychotics. In addition to medication, many forms of psychotherapy may help people with schizophrenia include: Comprehensive care programs are designed to incorporate therapeutic approaches on multiple levels, such
as:individualfamilyemployment educationcommunity participation A 2016 study found that comprehensive care programs may lead to greater success for schizophrenia have changed over time, cultural depictions and attitudes haven't caught up. Immense stigma around
schizophrenia still exists today, rooted in historical misconceptions and media depictions. The word "schizophrenic" might be used to define a person with the condition. Pervasive myths often falsely associate schizophrenia with: violencea "childlike" mind a "weak"
characterFor some people, the stigma around schizophrenia can lead to discomfort with a diagnosis. But as psychiatrist and author Elyn R. Saks wrote in her seminal 2007 book, "A mental illness diagnosis does not automatically sentence you to a bleak and painful life, devoid of pleasure or joy or accomplishment." Schizophrenia-like mental health
conditions have been recorded and treated since antiquity. Over centuries, theories about the condition's causes evolved from the spiritual realm to physiological means. Treatments for schizophrenia have often included inhumane and cruel "cures," even as recently as the 20th century. But thanks to evidence-based research and medical science,
modern physicians and therapists have a wealth of effective tools to help treat schizophrenia. If you are living with schizophrenia, you are not alone. And if someone you love is impacted by the condition, it may be helpful to learn about how to support your loved one. Angane AY. (2017). The divine madness: A history of schizophrenia.
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care for first episode psychosis: Two-year outcomes from the NIMH RAISE early treatment program J, et al. (1984). Sin and mental illness in the Middle Ages. T. (2021). Personal Interview.Mental health by the numbers. (2021). illness and violence: Debunking myths, addressing realities. (2021). shock treatment. (2020). R, et al. (2010). Examining
differences in the stigma of depression and schizophrenia. E. (2007). The Center Cannot hold: My Journey Through Madness. New York, New 
(2019). The Collected Schizophrenias. Minneapolis, Minnesota: Graywolf PressWhat is electroconvulsive therapy (ECT)? (2021). Less than 200 years ago schizophrenia emerged from a tangle of mental disorders known simply as madness. In the upcoming fifth edition of psychiatry's primary guidebook, the Diagnostic and Statistical Manual of Mental
Disorders, or DSM-5, schizophrenia will finally shed the outdated, 19th-century descriptions that have characterized it to this day. Yet the disorder remains poorly understood. "There is substantial dissatisfaction with schizophrenia treated as a disease entity; its symptoms are like a fever—something is wrong, but we don't know what," says William
Carpenter, a psychiatrist at the University of Maryland and chair of the manual's Psychotic Disorders Work Group. Psychiatrists may discover that this disorder is not a single syndrome after all but a bundle of related conditions. Madness, Demons and Delusionscirca 1550 B.C.The Book of Hearts from ancient Egypt records how poison, demons, fecal
matter or blood trouble may be at the root of madness. On supporting science journalism by subscribing a subscribing our world today.ca. 1400 B.C. The
ancient Hindu Vedas describe illnesses characterized by bizarre behavior, lack of self-control, filth and nudity brought on by devils.ca. 400 B.C.Another early text, The Yellow Emperor's Classic of Internal Medicine from China, describes how insanity, dementia and seizures arose from demonic possession and other supernatural forces.1334
A.D.Opicinus de Canistris, an Italian scribe, is struck by a strange illness in which he believes he sees the Virgin Mary. Afterward, he becomes socially withdrawn and devotes his time to creating fantastical maps. The case may describe either a stroke or an early example of schizophrenia. Early Dementia 1809 French and English physicians Philippe
Pinel and John Haslam independently describe young patients who show signs of "premature dementia." These cases are commonly referred to as the first thorough portraits of schizophrenia. 1834Ukrainian writer Nikolay Gogol's Diary of a Madman follows Poprishchin, whose delusions of grandeur accompany his slide into insanity. The story is
considered one of the earliest descriptions of schizophrenia in literature.1871German psychiatrists Karl Ludwig Kahlbaum describes catatonia—characterized by alternating immobility and frenzy—and paranoia, in which an individual is overwhelmed by fear. Hecker
iden-tifies hebephrenia, which involves erratic behavior and incoherent speech.1896Emil Kraepelin, a German psychiatrist, describes dementia praecox as a lifelong disease that worsens with time. He later incorporates Hecker and Kahlbaum's disorders as syndromes of dementia praecox.1906Swiss psychiatrist Adolf Meyer rejects Kraepelin's
concept of dementia praecox as a biological disease. Instead he favors a psychoanalytic approach, in which mental illness is triggered by life experiences, such as stress or a difficult childhood. Meyer's ideas will influence the DSM-I and DSM-II descriptions of schizophrenia. "Schizophrenia" 1908Swiss psychiatrist Eugen Bleuler, who did not believe
that the disorder known as dementia praecox always involved deterioration and only struck adolescents, coins the term "schizophrenia," referring to a split mind.1914First use of term "schizophrenia," referring to a split mind.1914First use of term "schizophrenia," referring to a split mind.1914First use of term "schizophrenia," referring to a split mind.1914First use of term "schizophrenia," referring to a split mind.1914First use of term "schizophrenia," referring to a split mind.1914First use of term "schizophrenia," referring to a split mind.1914First use of term "schizophrenia," referring to a split mind.1914First use of term "schizophrenia," referring to a split mind.1914First use of term "schizophrenia," referring to a split mind.1914First use of term "schizophrenia," referring to a split mind.1914First use of term "schizophrenia," referring to a split mind.1914First use of term "schizophrenia," referring to a split mind.1914First use of term "schizophrenia," referring to a split mind.1914First use of term "schizophrenia," referring to a split mind.1914First use of term "schizophrenia," referring to a split mind.1914First use of term "schizophrenia," referring to a split mind.1914First use of term "schizophrenia," referring to a split mind.1914First use of term "schizophrenia," referring to a split mind.1914First use of term "schizophrenia," referring to a split mind.1914First use of term "schizophrenia," referring to a split mind.1914First use of term "schizophrenia," referring to a split mind.1914First use of term "schizophrenia," referring to a split mind.1914First use of term "schizophrenia," referring to a split mind.1914First use of term "schizophrenia," referring to a split mind.1914First use of term "schizophrenia," referring to a split mind.1914First use of term "schizophrenia," referring to a split mind.1914First use of term "schizophrenia," referring to a split mind.1914First use of term "schizophrenia," referring to a split mind.1914First use of term "schizophrenia," referring to a split mind.
schizophrenia. She spends the rest of her life in mental hospitals. Her husband, F. Scott Fitzgerald, is inspired to write the novel Tender Is the Night about a woman's mental illness. 1958Premiere of Suddenly Last Summer, by Tennessee Williams, who
was diagnosed with schizophrenia and underwent a lobotomy.1959German psychiatrist Kurt Schneider identifies core symptoms of schizophrenia, such as auditory hallucinations and delusions, that distinguish it from other forms of psychosis. His criteria end up informing the DSM-III. Biological Mystery1972Neurologist Fred Plum calls the disorder a
 "graveyard of neuropathologists," a reference to the apparent lack of a brain-based explanation for schizophrenia. This is the first of many studies to identify brain anomalies associated with schizophrenia. 1980Building on Kraepelin's
definition, the DSM-III recognizes five subtypes of schizophrenia: disorganized (hebephrenic), catatonic, paranoid, residual and undifferentiated.1986German psychiatrist Karl Leonhard suggests schizophrenia is a group of psychoses—including hallucinations and cognitive dysfunction—rather than a single disorder.1994Mathematician John Forbes
Nash, Jr., receives a Nobel Prize. His struggles with schizophrenia are the subject of A Beautiful Mind, a biography by Sylvia Nasar and an eponymous Oscar-winning film.1994The DSM-IV is published. It includes subtypes of schizophrenia but notes their limited utility in diagnosis. Contemporary Questions 2002The Japanese Society of Psychiatry and
Neurology changed the name of schizophrenia from seishin bunretsu byo, or "mind-split disease," to togo shitcho sho, or "integration disorder." The change has reduced stigma and confusion about the nature of the disease. 2009In what New York Times reporter Nicholas Wade dubs "a Pearl Harbor of schizophrenia research," three studies in Nature
journals implicate between tens and thousands of possible gene variants. 2013The DSM-5 will remove the subtypes of schizophrenia, patients must now exhibit delusions, hallucinations or disorganized speech. They may also experience motor difficulties, such as catatonia, and negative symptoms, such as social
withdrawal or lack of emotional responsiveness. Get the MedicineNet Daily newsletter for health tips, wellness updates and more. By clicking "Subscribe Now", I agree to receive emails from MedicineNet and I understand that I may opt out of MedicineNet subscriptions at any
time. I have a family member with schizophrenia is not directly passed from one generation to another genetically, and there is no single specific cause for this illness. Rather, it is the
result of a complex group of genetic and other biological vulnerabilities, as well as psychological and environmental risk factors. Biologically, it is thought that people who have abnormalities in the brain neurochemical dopamine and lower brain issues that
are thought to predispose people to developing schizophrenia include abnormalities in the connectivity. Recent research is emerging that implicates potential abnormalities in the transmission of the brain neurochemical glutamate as a risk factor for having schizophrenia.
Schizophrenia is thought to have a significant but not solely genetic component. Genetically, schizophrenia and bipolar disorders share a number of the same risk genes. However, the fact is that both illnesses also have some genetic factors that are unique. There are some genetic commonalities with
schizophrenia and epilepsy, as well. Environmentally, the risks of developing schizophrenia is increased in individuals whose father is of advanced age or whose mother was malnourished or had one of certain infections during pregnancy. Difficult life circumstances during childhood,
like the early loss of a parent, parental poverty, bullying, witnessing domestic violence; being the victim of emotional, sexual, or physical abuse or of physical or emotional neglect; and insecure attachment have been associated with increased risks of developing this illness. Using drugs, particularly marijuana (cannabis), amphetamines, and
hallucinogens, has been found to increase the risk of developing schizophrenia. Factors like recent migration, being discriminated against, and how well represented an ethnic group is in a neighborhood can also be a risk or protective factor for developing schizophrenia. For example, some research indicates that ethnic minorities may be more at risk
for developing this disorder if there are fewer members of the ethnic group to which the individual belongs in their neighborhood. Read our full medical article to learn more about schizophrenia signs, symptoms, treatment, and prognosis. CONTINUE SCROLLING OR CLICK HERE As a library, NLM provides access to scientific literature. Inclusion in
an NLM database does not imply endorsement of, or agreement with, the contents by NLM or the National Institutes of Health. Learn more: PMC Disclaimer | PMC Copyright Notice More than a century since the delineation of dementia praecox by Kraepelin, the etiology, neuropathology and pathophysiology of schizophrenia remain elusive. Despite
the availability of criteria allowing reliable diagnostic identification, schizophrenia essentially remains a broad clinical syndrome defined by reported subjective experiences (symptoms), loss of function (behavioral impairments), and variable patterns of course. Research has identified a number of putative biological markers associated with the
disorder, including neurocognitive dysfunction, brain dysmorphology, and neurochemical abnormalities. Yet none of these variables has to date been definitively proven to possess the sensitivity and specificity expected of a diagnostic test. Genetic linkage and association studies have targeted multiple candidate loci and genes, but failed to
demonstrate that any specific gene variant, or a combination of genes, is either necessary or sufficient to cause schizophrenia. Thus, the existence of a specific brain disease underlying schizophrenia remains a hypothesis. Against a background of an ever-increasing volume of research data, the inconclusiveness of the search for causes of the disorder
the host of clinical phenomena and research data consistent with a disease hypothesis of schizophrenia. For the time being, the clinical concept of schizophrenia is supported by empirical evidence that its multiple facets form a broad syndrome with non-negligible internal cohesion and a characteristic evolution over time. The dissection of the
syndrome with the aid of endophenotypes is beginning to be perceived as a promising approach in schizophrenia genetics. Keywords: nosology, category, dimension, validity, utility, endophenotypes is beginning to be perceived as a promising approach in schizophrenia genetics. Keywords: nosology, category, dimension, validity, utility, endophenotypes is beginning to be perceived as a promising approach in schizophrenia genetics. Keywords: nosology, category, dimension, validity, utility, endophenotypes is beginning to be perceived as a promising approach in schizophrenia genetics.
escurridizas. A pesar de la disponibilidad de criterios que permiten identificaciones diagnôsticas confiables, la esquizofrenia sigue siendo esencialmente un amplio sfindrome clfnico definido por el relato de experiencias subjetivas (sfintomas), la pérdida del funcionamiento (deterioros conductuales) y patrones variables de evolución. La investigación ha
identificado un núméro de reputados marcadores biolôgicos asociados con el trastorno, incluyendo disfunciôn neurocognitiva, alteraciones de la morfologfa cerebral y anormalidades neuroqufmicas. A la fecha todavfa ninguna de estas variables ha demostrado que posea definitivamente la sensibilidad y especificidad esperadas para una prueba
diagnôstica. Los estudios genéticos de ligamiento y de asociaciôn han apuntado a múltiples loci y genes candidato, pero no se ha podido demostrar que alguna variante específica de un gen, o de una combinaciôn de genes, sea necesaria o suficiente para causar la esquizofrenia. Por lo tanto, la existencia de una enfermedad cerebral específica a la
base de la esquizofrenia sigue constituyendo una hipôtesis. En oposición a los antécédentes de un volumen siempre creciente de información proveniente de la investigación, la falta de conclusiones en la bûsqueda de causas para este trastorno genera dudas acerca de la validez del constructo esquizofrenia como está actualmente definido
Considerando la naturaleza versâtil de los sfntomas de la esquizofrenia y la pobre coherencia de los fenômenos clfnicos y biolôgicos, taies dudas son razonables. Sin embargo, el desmantelar simplemente el concepto es poco probable que se traduzca en un modelo alternativo que pudiera dar cuenta de la presentación de los fenômenos clfnicos y de los
datos de la investigación que sean consistentes con la hipôtesis de enfermedad de la esquizofrenia. Por ahora, el concepto clfnico de la esquizofrenia está sustentado por la evidencia empfrica en que sus múltiples presentaciones forman un amplio sfindrome con una coherencia interna no insignificante y una evolución caracterfstica a lo largo del
tiempo. La disección del sfndrome con la ayuda de endofenotipos está comenzando a ser percibida como una prometedora aproximación en la genética de la esquizofrenia. Plus d'un siècle après la description de la démence précoce par Kraepelin, l'étiologie, la neuropathologie et la physiopathologie de la schizophrénie demeurent difficiles à
appréhender. Malgré la disponibilité de critères permettant une identification diagnostique fiable, la schizophrénie reste surtout un vaste syndrome clinique défini par le rapport d'expériences subjectives (symptômes), la perte d'une fonction (déficits comportementaux) et des modalités évolutives variables. La recherche a trouvé plusieurs marqueurs marqu
biologiques possibles associés à la maladie comme des dysfonctions neurocognitives, des anomalies morphologiques cérébrales et des désordres neurochimiques. Aucune de ces variables n'a encore montré à ce jour la sensibilité et la spécificité attendues d'un test diagnostique. Les études d'association et de liaison génétiques ont ciblé de nombreux
gènes et locus candidats sans mettre en évidence un variant de gène spécifique ou une combinaison de gènes nécessaire ou suffisant pour provoquer la schizophrénie. L'existence d'une maladie cérébrale spécifique sous-tendant la schizophrénie reste donc une hypothèse. Les données de la recherche sont en augmentation constante mais leur
incapacité à conclure sur les causes de la schizophrénie fait douter de la validité de sa définition actuelle. Et ces doutes trouvent leur source dans la nature inconstante des symptômes de la schizophrénie et la faible cohérence des résultats cliniques et biologiques. Cependant, un simple démantèlement du concept ne suffira pas à produire un modèle
alternatif capable d'expliquer la série de symptômes cliniques et des données de recherche concordant avec une hypothèse de maladie pour la schizophrénie. Pour l'instant, le concept cliniques et des données de recherche concordant avec une hypothèse de maladie pour la schizophrénie.
évolution caractéristique dans le temps. L'analyse fine de ce syndrome à l'aide d'endophénotypes commence à être perçue comme une approche prometteuse dans la génétique de la schizophrénie. To the best of present knowledge, schizophrenia is a disorder with variable phenotypic expression and poorly understood, complex etiology, involving a
major genetic contribution, as well as environmental factors interacting with the genetic susceptibility. Multiple genes and different combinations of their polymorphic variants provide the genetic background, with a proportion of the transmitted genetic susceptibility. Multiple genes and different combinations of their polymorphic variants provide the genetic susceptibility.
rates, 1 which is consistent with an ancient origin and - as far as records go - its incidence has not changed much over the past two centuries. Diagnostic concepts play a critical role in the management and treatment of schizophrenia patients; in research aiming to identify risk factors and causal mechanisms, as well as in attempts to resolve
contentious issues, such as comorbidity and relationships among proximal or partly overlapping disorders. A principal source of difficulty in this endeavor is the complex nature of the disorder itself, and the inherent weakness of the diagnostic concept of schizophrenia, in that it remains based upon assumptions about an underlying but still unknown
disease process. Most of the attributes defining schizophrenia are primarily inferential and depend on self-reported subjective experience. The underlying structural and functional pathology is insufficiently understood, and there is no objective experience. The underlying structural and functional pathology is insufficiently understood, and there is no objective experience.
making or biological and epidemiological research. Recurrent controversies in schizophrenia research concern its delimitation from other psychoses, bipolar affective disorder, and neurodevelopmental disorders; the utility of its
categorical classification as compared with descriptive symptom dimensions or subtypes based on quantitative cognitive traits,2 and the discordances between the ICD-10 and DSM-IV criteria for its diagnostic concept of schizophrenia
ending with a speculation about its future prospects. The disease concept of schizophrenia is of a relatively recent origin, as compared with disorders such as melancholia, mania, or generic "insanity," all known since antiquity. By the middle of the 19th century, European psychiatrists began describing disorders of unknown causes, typically affecting
the young, and often progressing to chronic deterioration. In France, Morel3 referred to such cases as démence précoce, while in Scotland, Clouston4 coined the term "adolescent insanity." In Germany, Kahlbaum5 delineated the catatonic syndrome, and his disciple Hecker6 described hebephrenia. However, it was Emil Kraepelin (1856-1926) who
proposed to integrate those varied clinical pictures into a single nosological entity under the name of "dementia praecox," based on his longitudinal observations of a large number of clinical cases exhibiting a common pattern of course which ultimately resulted in severe cognitive and behavioral decline. Elaborating on the description of the disorder
 in successive editions of his Textbook,7,8 Kraepelin acknowledged the diversity of the clinical pictures subsumed under dementia praecox and articulated nine different "clinical forms" (Table I). Although the core features of the disorder could not always be identified reliably in the cross-section of the clinical presentation, Kraepelin emphasised that
 "we meet everywhere the same fundamental disorders in the different forms of dementia praecox [...] in very varied conjunctions, even though the clinical picture may appear at first sight ever so divergent. 8 The "fundamental disorders" which supported the concept of the disease entity were cognitive deficit (a "general decay of mental efficiency")
and executive dysfunction ("loss of mastery over volitional action"), most clearly manifested in the residual, "terminal states" of the illness. Kraepelin was reluctant to impute etiological significance to the clinical variants he described, and regarded the issue of a unitary process versus multiple disease states within dementia praecox "an open
question." His approach to the definition and classification of psychiatric disorders was, essentially, based on comprehensive clinical observations and naturalistic descriptions of a large number of individual cases. Kraepelin never issued a definitive list of diagnostic criteria for dementia praecox and was particularly careful to avoid claims about any
 "pathognomonic" symptoms. The ultimate validation of the disease entity, Kraepelin believed, would come from neuropathology, physiology, and biological chemistry of the brain, whereas the specific contribution of clinical research consisted in identifying replicable patterns of intercorrelations between symptoms, course, and outcome.
 praecox simplex ("Impoverishment and devastation of the whole psychic life which is accomplished quite imperceptibly") • Hebephrenia (Insidious change of personality with shallow capricious affect, senseless and incoherent behaviour, poverty of thought, occasional hallucinations and fragmentary delusions, progressing to profound dementia)
 Depressive dementia praecox (simple and delusional form) (Initial state of depression followed by slowly progressive cognitive decline and avolition, with or without hypochondriacal or persecutory delusions, marked fluctuations of
mood and aimless impulsivity) • Agitated dementia praecox (Recurrent acute, brief episodes of confused excitement with remissions) • Catatonia ("Conjunction of peculiar excitement with catatonic stupor dominates the clinical picture"
in this form, but catatonic phenomena frequently occur in otherwise wholly different presentations of dementia praecox) • Paranoid dementia frequently occur in otherwise wholly different presentations and hallucinations. The severe form results in a "peculiar disintegration of psychic life", involving especially emotional and volitional disorders.
The mild form is a very slowly evolving "paranoid or hallucinatory weak-mindedness" which "makes it possible for the patient for a long time still to live as an apparently healthy individual") • Schizophasia (confusional speech dementia praecox) (Cases meeting the general description of dementia praecox but resulting in an end state of "an unusually individual") • Schizophasia (confusional speech dementia praecox) (Cases meeting the general description of dementia praecox but resulting in an end state of "an unusually individual") • Schizophasia (confusional speech dementia praecox) (Cases meeting the general description of dementia praecox) (Cases meeting t
striking disorder of expression in speech, with relatively little impairment of the remaining psychic activities") Kraepelinis views on the typology of mental disorders - often quoted, occasionally misquoted, and still debated - continue to frame much of the present-day psychiatric discourse. It looks indeed as if "psychiatry still lives in a Kraepelinian
world," 9 but the exact contours of its map often get blurred. Towards the end of his career Kraepelin experienced doubts about the validity of his original formulation of the problem may be incorrect." 10 He considered abandoning the
 categorical disease notions of schizophrenia and manic-depressive disorder, and replacing them with a sort of dimensional model in which schizophrenic and affective syndromes "do not represent the expression of particular pathological processes, but rather indicate the areas of our personality in which these processes unfold." 10 The role of
 "hereditary factors" was to "make certain areas more susceptible and accessible to pathological stimuli." According to Kraepelin, "the various syndromes of illness may be compared with the different registers of an organ, any of which may be brought into play according to the severity or extent of the pathological changes involved. They impart a
characteristic tone to the illness quite irrespective of the mechanism which has brought them into play." He introduced a notion of phylogenetically preformed templates of brain responses that could be released by a variety of morbid processes - an idea with obvious links to Hughlings Jackson's theory of the dissolution of higher cortical functions.11
Kraepelin proposed three hierarchically structured "registers" of psychopathology - affective, schizophrenic, and encephalopathic - which could recombine in different ways to produce the manifold syndromes of the major mental disordres. Eugen Bleuler (1857-1939) significantly modified Kraepelin's original concept by adding to its scope clinical
illnesses which did not evolve into the kind of "terminal state" of deterioration, considered by Kraepelin to be the hallmark of the disease. Having coined the term "schizophrenia" to replace dementia praecox, Bleuler12 stated that schizophrenia "is not a disease in the strict sense, but appears to be a group of diseases [ ...] Therefore we should speak
of schizophrenias in the plural." Importantly, Bleuler introduced a fundamental distinction between basic (obligatory) and accessory symptoms of the disorder. While the accessory symptoms included thoughten basic symptoms of the disorder. While the accessory symptoms of the disorder.
and speech derailment ("loosening of associations"), volitional indeterminacy ("ambivalence"), affective incongruence, and withdrawal from reality ("autism"). It was the presence of the basic symptoms that, according to Bleuler, gave schizophrenia its distinctive diagnostic profile. He acknowledged that the clinical subgroups of paranoid
schizophrenia, catatonia, hebephrenia, and simple schizophrenia were not "natural" nosological entities and argued that "schizophrenia must be a much broader concept than the overt psychosis of the same name." Along with the "latent" schizophrenias, which presented attenuated forms of the basic symptoms, manifesting as aberrant personality
traits, he also listed within the "broader concept" atypical depressive or manic states, Wernicke's motility psychoses, and other nonorganic, nonaffective psychoses, and other nonorganic, nonaffective psychoses, reactive psychoses, and other nonorganic, nonaffective psychoses, and other nonorganic, nonaffective psychoses, reactive psychoses, and other nonorganic, nonaffective psychoses, reactive psychoses, and other nonorganic, nonaffective psychoses, and other nonorganic psychoses, and other no
spectrum disorders. During the ensuing decades, a number of European and American clinicians proposed further subnosological distinctions within the widening phenotype of schizophrenia, including schizophrenia, including schizophrenia distinctions within the widening phenotype of schizophrenia distinctions within the widening phenotype distinction distinction distinction distinction distinction distinction distinction dist
that nine groups of psychotic manifestations, designated as "firstrank symptoms" (FRS), had a "decisive weight" in the diagnosis of schizophrenia: audible thoughts; voices arguing about, or discussing, the patient; voices commenting on the patient's actions; experiences of influences on the body; thought withdrawal and other interference with
thought; thought broadcast (diffusion of thought); delusional perception; and other experiences involving "made" impulses and feelings experienced as caused by an outside agency. Due to the sharpness of their definition and the hope that they could be reliably ascertained, the FRS were subsequently incorporated in the Research Diagnostic Criteria
RDC,18 DSM-III,19 and ICD-10.20 The Catego algorithm,21 used in the WHO cross-national studies, defined a "nuclear" schizophrenia (S+) characterized by presence of at least 3 out of 6 FRS. Familiality and modest to substantial heritability has been reported for the FRS,22 but a recent study23 found that these symptoms did not predict severee.
deterioration and cognitive deficit in schizophrenia patients. In a clinical tradition of the "endogenous" psychoses which departed substantially from the Kraepelinian and Bleulerian nosology. Leonhard
defined sharply delineated disease entities, described by a detailed psychopathology emphasizing objective signs (eg, psychomotor behavior), course and outcome, and family history. The nonaffective psychoses were split into "systematic" and "unsystematic" and "unsystematic" groups of schizophrenias, and a third group of "cycloid" psychoses, each containing further
subtypes (Table II), for which Leonhard claimed distinct categorical disease status. While the "unsystematic" schizophrenias were considered to be primarily genetic, hereditary factors were thought to play a secondary role in the cycloid psychoses and the "systematic" schizophrenias, which were presumed to be exogenously determined, eg, by
maternal obstetric complications or early failure of social learning. Notably, Leonhard's classification neither expands, nor constricts, the outer boundaries of schizophrenia spectrum in a different way. I. Group of systematic schizophrenia spectrum in a different way. I. Group of systematic schizophrenia spectrum in a different way. I. Group of systematic schizophrenia spectrum in a different way. I. Group of systematic schizophrenia spectrum in a different way. I. Group of systematic schizophrenia spectrum in a different way. I. Group of systematic schizophrenia spectrum in a different way. I. Group of systematic schizophrenia spectrum in a different way. I. Group of systematic schizophrenia spectrum in a different way. I. Group of systematic schizophrenia spectrum in a different way. I. Group of systematic schizophrenia spectrum in a different way. I. Group of systematic schizophrenia spectrum in a different way. I. Group of systematic schizophrenia spectrum in a different way. I. Group of systematic schizophrenia spectrum in a different way. I. Group of systematic schizophrenia spectrum in a different way. I. Group of systematic schizophrenia spectrum in a different way. I. Group of systematic schizophrenia spectrum in a different way. I. Group of systematic schizophrenia spectrum in a different way. I. Group of systematic schizophrenia spectrum in a different way. I. Group of systematic schizophrenia spectrum in a different way. I. Group of systematic schizophrenia spectrum in a different way. I. Group of systematic schizophrenia spectrum in a different way. I. Group of systematic schizophrenia spectrum in a different way. I. Group of systematic schizophrenia spectrum in a different way. I. Group of systematic schizophrenia spectrum in a different way. I. Group of systematic schizophrenia spectrum in a different way. I. Group of systematic schizophrenia spectrum in a different way. I. Group of systematic schizophrenia spectrum in a different way. In the systematic schizophrenia spectrum in a different way
of affect, continuous unremitting course, personality deterioration) Paraphrenias (Auditory hallucinosis, audible thoughts, thought broadcast, passivity experiences, delusional misidentifications of memory) Hebephrenias (Excessivences, delusional misidentifications, falsifications, falsifications, falsifications) Paraphrenias (Excessivences, delusional misidentifications, falsifications) Paraphrenias (Excessivences, delusional misidentifications) Paraphrenias (Excessivences, delusi
parakinesias, mannerisms, verbigeration, posturing, stereotypies, mutism, auditory hallucinations) II. Group of unsystematic (atypical) schizophrenias (Rapid onset, relatively preserved affect, remitting course, mild personality deterioration) Affect-laden paraphrenia (Paranoid delusions with affective loading) Cataphasia (schizophasia) (Incoherent
pressured speech but well-organised behaviour) Periodic catatonia (Episodic hyper- or hypokinesia, mixed excitatory and hallucinatory symptoms) III. Group of cycloid psychoses (Sudden onset, pervasive delusional mood, multimodal hallucinatory symptoms) III. Group of cycloid psychoses (Sudden onset, pervasive delusional mood, multimodal hallucinatory symptoms) III.
psychosis (Extreme shifts of affect, polarity intense fear - ecstatic elation) Motility psychosis (Impulsive hypermotility - psychomotor inhibition) Confusion psychosis (Incoherent pressure of speech - mutism) The concept of a continuum or spectrum of schizophrenia-related phenotypes originates in the observation that several ostensibly different
disorders tend to cluster among biological relatives of individuals with clinical schizophrenia.25 Epidemiological and family studies suggest that the genetic liability to schizophrenia is shared with liability to other related syndromes.26,27 The term "schizotypy," first introduced by Rado28 and Meehl,29 describes a personality characterized by
anhedonia, ambivalence, "interpersonal aversiveness," body image distortion, "cognitive slippage, "and sensory, kinesthetic, or vestibular aberrations and "magical ideation" as traits predicting "psychosis proneness." These constructs were later amalgamated with clinical
descriptions from the Danish-US adoptive study into the DSM-III diagnostic category of schizotypal personality disorder (SPD), which is now central to the spectrum notion.31 The frequent occurrence of SPD among first-degree relatives of probands with schizophrenia has been replicated in the Roscommon epidemiological study,32 which added to
the schizophrenia spectrum further disorders (ii) schizophrenia; (ii) schizophrenia; (iii) schizophrenia; (iii) schizophrenia; (iii) schizophrenia; (iii) schizophrenia; (iii) schizophrenia; (iii) schizophrenia; (iv) other nonaffective psychotic disorders; (iv) other nonaffective psychotic d
affective disorders. In all its variations, the spectrum concept remains critically dependent on the validity of the SPD concept. Accumulating evidence from family and twin data indicates that SPD is multidimensional and may be genetically heterogeneous.33,35 Its manifestations fall into two genetically independent clusters: a "negative" cluster (odd
speech and behavior, inappropriate affect, and social withdrawal), more common among relatives of schizophrenic probands, and a "positive" cluster (magical ideation, brief quasipsychotic episodes), associated with increased incidence of affective disorders in relatives. "Negative" schizotypy may indeed represent a subclinical forme fruste of
schizophrenia, manifesting attenuated cognitive deficits and brain structural abnormalities. A general "weakening" of mental processes resulting in a "defect" was the cornerstone of Kraepelin's dementia praecox, who suggested that precursors of "defect" was the cornerstone of Kraepelin's dementia praecox, who suggested that precursors of "defect" was the cornerstone of Kraepelin's dementia praecox, who suggested that precursors of "defect" was the cornerstone of Kraepelin's dementia praecox, who suggested that precursors of "defect" was the cornerstone of Kraepelin's dementia praecox, who suggested that precursors of "defect" was the cornerstone of Kraepelin's dementia praecox, who suggested that precursors of "defect" was the cornerstone of Kraepelin's dementia praecox, who suggested that precursors of "defect" was the cornerstone of Kraepelin's dementia praecox, who suggested that precursors of "defect" was the cornerstone of Kraepelin's dementia praecox, who suggested that precursors of "defect" was the cornerstone of Kraepelin's dementia praecox, who suggested that precursors of "defect" was the cornerstone of Kraepelin's dementia praecox, who suggested that precursors of "defect" was the cornerstone of Kraepelin's dementia praecox, who suggested that precursors of "defect" was the cornerstone of Kraepelin's dementia praecox, who suggested that precursors of "defect" was the corners of the
the 1970s, the terms "defect" and "productive" symptoms have been virtually replaced by "negative" and "positive" symptoms." 36 Crow37 proposed a simple subclassification of schizophrenia, based on the predominance of either positive or negative symptoms have been virtually replaced by "negative" symptoms." 36 Crow37 proposed a simple subclassification of schizophrenia, based on the predominance of either positive or negative symptoms."
and formal thought disorder, with a presumed underlying dopaminergic dysfunction, while patients with "Type II" (negative) schizophrenia displayed social withdrawal, loss of volition, affective flattening, and poverty of speech, presumed to be associated with structural brain abnormalities. Criteria and rating scales for positive (SAPS) and negative
(SANS) schizophrenia were proposed by Andreasen and Olsen.38 The initial typology, implying discrete, mutually exclusive "types," was later replaced by a negative and a positive dimension, allowing the two kinds of symptoms to co-occur in the same individual.39 Carpenter and collaborators 40,41 proposed the delineation of a subtype of
schizophrenia characterized by enduring "primary" negative symptoms that could not be construed as sequelae of other psych opathology (Table III). This clinical construct, evocative of Kraepelin's dementia praecox, was termed "deficit schizophrenia" (DS) and hypothesized to be an etiologically distinct "disease" within the schizophrenia
spectrum.42 Studies comparing DS cases with "nondeficit" (NDS) patients and controls, estimated the prevalence of the DS subtype at 16.5% in unselected epidemiological samples of schizophrenia cases and 25% to 30% within samples of chronic schizophrenia. DS and NDS do not differ on age at onset and length of illness, which argues against a
progression leading from NDS to DS. Limited support for the DS construct has been provided by neuropsychological studies and assessment of soft neurological studies and assessment of soft neurologic
negative symptoms must be present: Restricted affect Diminished emotional range Poverty of speech Curbing of interests Diminished sense of purpose Diminished sense Diminished sense of purpose Diminished sense of purpose Dimini
negative symptoms are primary, ie, not secondary to factors other than the disease process, eg, Anxiety Drug effects Suspiciousness or other psychotic symptoms Mental retardation Depression 4. The patient meets DSM-III criteria for schizophrenia Factor analysis and related methods reduce any correlations present within the data matrix to
covariances of a small number of latent factors which account for the interrelationships among the primary variables (SANS/SAPS scores), a three-factor structure has been proposed 47 and subsequently replicated. 48-50 In this model, negative symptoms
strongly on the content of the input - studies using SANS and SAPS result in different solutions from those based on scale (BPRS), or Operational Criteria Checklist (OPCRIT). In a large sample of schizophrenia probands, McGrath et al53 identified 5 factors
(positive, negative, disorganized, affective, and early onset/developmental) associated with risk of psychoses and affective disorders in relatives. In a series of factor analyses based on an expanded list of 64 psychoses and affective disorders in relatives. In a series of factor analyses based on an expanded list of 64 psychoses and affective, and early onset/developmental)
of schizophrenia should be viewed with caution, considering the diversity of clinical populations and the limitations of the instruments used to generate the input data. Whereas factor analysis groups variables, cluster analysis groups individuals on the basis of maximum shared characteristics. Farmer et al55 identified two clusters into which patients
with schizophrenia could be fitted, based on scores of 20 symptom and history items: one characterized by good premorbid functioning, incoherent speech, bizarre behavior, and family history of schizophrenia. Using PANSS, Dollfus et al56
obtained 4 quite different clusters, corresponding to positive, negative, disorganized, and mixed symptomatology. Thus, cluster analysis is as dependent on the selection of input variables as factor analysis. Latent class analysis (LCA) assumes the existence of a finite number of mutually exclusive and jointly exhaustive groups of individuals. A latent
class typology of schizophrenia, proposed by Sham et al,57 using data on 447 patients with nonaffective psychoses, suggested three subgroups: a "neurodevelopmental" subtype resembling the hebephrenic form of the disorder (poor premorbid adjustment, early onset, prominent negative and disorganized features); a "paranoid" subtype (less severe,
 better outcome); and a "schizoaffective" subtype (dysphoric symptoms). In an epidemiological sample of 343 probands with schizophrenia and affective disorders, Kendler et al58 found 6 latent classes, broadly corresponding to the nosological forms of "Kraepelinian" schizophrenia: major depression, schizophreniform disorder, schizoaffective disorders, Kendler et al58 found 6 latent classes, broadly corresponding to the nosological forms of "Kraepelinian" schizophrenia: major depression, schizophrenia and affective disorder, schizophrenia and affective disorders, Kendler et al58 found 6 latent classes, broadly corresponding to the nosological forms of "Kraepelinian" schizophrenia and affective disorders, Kendler et al58 found 6 latent classes, broadly corresponding to the nosological forms of "Kraepelinian" schizophrenia and affective disorders, Kendler et al58 found 6 latent classes, broadly corresponding to the nosological forms of "Kraepelinian" schizophrenia and affective disorders, Kendler et al58 found 6 latent classes, broadly corresponding to the nosological forms of "Kraepelinian" schizophrenia and affective disorders, kendler et al58 found 6 latent classes, broadly corresponding to the nosological forms of "Kraepelinian" schizophrenia and affective disorders and affective disorders are also affective disorders.
(manic), schizoaffective disorder (depressed), and hebephrenia. Similar results, using a combination of principal component analysis and LCA in an epidemiologically ascertained sample of 387 patients with psychoses have been reported by Murray et al.59 In contrast to conventional LCA, a form of latent structure analysis, known as grade of
membership (GoM), allows individuals to be members of more than one disease class and represents the latent groups as "fuzzy sets." 60,61 The GoM model simultaneously extracts from the data matrix a number of latent groups as "fuzzy sets." 60,61 The GoM model simultaneously extracts from the data matrix a number of latent groups as "fuzzy sets." 60,61 The GoM model simultaneously extracts from the data matrix and assigns to each individual a set of numerical weights quantifying the degree to which that individual resembles
each one of the identified pure types. When applied to the symptom profiles of 1065 cases in the WHO International Pilot Study of Schizophrenia, 2 to affective disorders, and 1 to patients in remission, all showing significant associations with course and outcome variables
used as external validators. Subtyping schizophrenia by the presence/absence of a positive family history for schizophrenia spectrum disorders was proposed as a strategy expected to be more successful in resolving heterogeneity than symptombased typologies.63 Familial (F) cases are usually defined as having >1 affected first-degree relative, while
sporadic (S) cases have no affected first- or second-degree relatives. The F/S dichotomy rests on the assumption that familial aggregation is primarily of a genetic origin, while sporadic cases result from environmental insults (eg, maternal obstetric complications) or de novo somatic mutations. In the majority of studies using this classification, the
proportion of familial cases was in the range of 8% to 15%. Since the F/S subtypes were hypothesized to differ etiologically, a number of studies, mostly of small to moderate sample size (50% of schizophrenia patients, 98 and there is compelling evidence that cognitive deficits are significantly correlated with impairments in activities of daily living
(ADL),99,100 but only weakly associated with psychotic symptoms.101 Population-based cohort studies102,103 have found that compromised general cognitive ability in late adolescence is a strong predictor of subsequent schizophrenia risk. Family studies indicate that a proportion of the unaffected first-degree relatives of index cases of
schizophrenia display similar patterns of deficit in an attenuated form.104-106 The balance of evidence suggests that cognitive dysfunction meets most of the criteria of an endophenotype in schizophrenia. This is underscored by the meta-analysis by Heinrichs and Zakzanis107 of 204 studies published between 1980 and 1994 (a total of 7420 schizophrenia).
schizophrenia patients and 5865 controls), in which effect sizes (Cohen's d) and the U statistic (degree of non-overlap) were calculated for 22 neurocognitive test variables ranging from IQ, verbal memory, and attention to executive function and language. Although no single test or cognitive construct was capable of separating perfectly schizophrenia
patients from normal controls, 7 measures achieved effect sizes greater than 1.0 (6070% non-overlap between the cases and controls): verbal memory (1.15), the Stroop task (1.11), and WAIS-R IQ (1.10). Although a subset of ~50% of
patients had nearly normal performance, significant cognitive impairment was common in schizophrenia and exceeded the deficits found in some neurological disorder that manifests itself in behavior. 107 There is, at least, a preliminary evidence that composite cognitive
endophenotypes have the capacity to identify genetically distinct subtypes of schizophrenia 108 More than a century since the delineation of dementia praecox by Kraepelin, the etiology, neuropathology, and pathophysiology of schizophrenia remain elusive. Despite the availability of criteria allowing reliable diagnostic identification, schizophrenia
essentially remains a broad clinical syndrome defined by reported subjective experiences (symptoms), loss of function (behavioral impairments) and variable patterns of course. Research has identified a number of putative biological markers associated with the disorder, including neurocognitive dysfunction, brain dysmorphology, and neurochemical
abnormalities. Yet none of these variables has to date been definitively proven to possess the sensitivity and specificity expected of a diagnostic test. Genetic linkage and association studies have targeted multiple candidate loci and genes, but failed to demonstrate that any specific gene variant, or a combination of genes, is either necessary or
sufficient to cause schizophrenia. Thus, the existence of a specific brain disease underlying schizophrenia remains a hypothesis. Against a background of an ever-increasing volume of research data, the inconclusiveness of the search for causes of the disorder fuels doubts about the validity of the schizophrenia construct as presently defined, some
leading to proposals to discard the category, 109 or to replace it with a continuum of "psychosis." 110 Given the protean nature of the symptoms of schizophrenia and the poor coherence of the clinical and biological findings, such doubts are not without reason. However, simply dismantling the concept is unlikely to result in an alternative model that
would account for the host of clinical phenomena and research data consistent with a disease hypothesis of schizophrenia is not a homogeneous entity, this has never been directly demonstrated, mainly because few studies of the appropriate kind have ever been undertaken. For the
time being, the clinical concept of schizophrenia is supported by empirical evidence that its multiple facets form a broad syndrome with non-negligible internal cohesion and a characteristic evolution over time. The dissection of the syndrome with non-negligible internal cohesion and a characteristic evolution over time.
genetics. As new concepts and data emerge from molecular genetics, cognitive science, or brain imaging, new perspectives on disease causation and brain function are likely to be on stage in the next decade. A recent strategic proposal about a future typology of psychiatric disorders, linking genomics and neural circuits functioning as "hubs" for a
range of phenotypes - cutting across the present categories and joining schizophrenia, autism, bipolar disorder, as well as forms of epilepsy and intellectual disability - may be a signpost of future developments.111 Such research must be supported by a refined, reliable, and valid phenotyping - not only at the level of symptoms, but increasingly
involving correlated neurobiological features. The study of endophenotypes transcending the conventional diagnostic boundaries may reveal unexpected patterns of associations with symptoms, personality traits, or behavior. The mapping of clinical phenomenology on specific brain dysfunction is now becoming feasible and the resulting functional
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E, et al. The future of psychiatric research: genomes and neural circuits. Science. 2010;327:1580-1581. doi: 10.1126/science.1188654. [DOI] [PMC free article] [PubMed] [Google Scholar] Articles from Dialogues in Clinical Neuroscience are provided here courtesy of Taylor & Francis The word schizophrenia was coined by the Swiss psychiatrist
Eugen Bleuler in 1908, and was intended to describe the separation of function between personality, thinking, memory, and perception. Bleuler introduced the term on 24 April 1908 in a lecture given at a psychiatric conference in Berlin and in a publication that same year.[1][2] Bleuler later expanded his new disease concept into a monograph in
1911, which was finally translated into English in 1950.[3][4] According to some scholars, the disease has always existed only to be 'discovered' during the early 20th century. The plausibility of this claim depends upon the success of retrospectively diagnosing earlier cases of madness as 'schizophrenia'. According to others, 'schizophrenia' names a
culturally determined clustering of mental symptoms. [5] What is known for sure is that by the turn of the 20th century the old concept of insanity and epilepsy (Emil Kraepelin's classification). [7] Dementia praecox was reconstituted as
schizophrenia, paranoia was renamed as delusional disorder and manic-depressive insanity as bipolar disorder (epilepsy was transferred from psychiatry to neurology). The 'mental symptoms' included under the concept schizophrenia are real enough, affect people, and will always need understanding and treatment.[citation needed] However,
whether the historical construct currently called 'schizophrenia' is required to achieve this therapeutic goal remains contentious. Accounts of a schizophrenia-like syndrome are thought to be rare in the historical record prior to the 19th century, although reports of irrational, unintelligible, or uncontrolled behavior were common.[8] There has been an
interpretation that brief notes in the Ancient Egyptian Ebers papyrus may imply schizophrenia,[9] but other reviews have not suggested any connection.[10] A review of ancient Greek and Roman literature indicated that although psychosis was described, there was no account of a condition meeting the criteria for schizophrenia.[11] Bizarre psychotic
beliefs and behaviors similar to some of the symptoms of schizophrenia were reported in Arabic medical and psychological literature during the symptoms of schizophrenia which he called Junun Mufrit (severe madness), which he
distinguished from other forms of madness (Junun) such as mania, rabies and manic depressive psychosis, [12] However, no condition resembling schizophrenia was reported in Serafeddin Sabuncuoğlu's Imperial Surgery, a major Ottoman medical textbook of the 15th century, [13] It has been suggested that the visions experienced by Joan of Arc were
the product of schizophrenia.[14] Given limited historical evidence, schizophrenia (as prevalent as it is today) may be a modern phenomenon, or alternatively it may have been obscured in historical writings by related concepts such as melancholia or mania.[8] Main article: Dementia praecox A detailed case report in 1809 by John Haslam concerning
James Tilly Matthews,[15] and a separate account by Philippe Pinel also published in 1809, are often regarded as the earliest cases of schizophrenia in the medical and psychiatry in 1886 in a textbook by asylum physician Heinrich Schüle (1840-1916) of the Illenau asylum in
 Baden. He used the term to refer to hereditarily predisposed individuals who were "wrecked on the cliffs of puberty" and developed acute dementia, while others developed the chronic condition of hebephrenia. Emil Kraepelin had cited Schüle's 1886 textbook in the 1887 second edition of his own textbook, Psychiatrie, and hence was familiar with
this term at least six years before he himself adopted it.[16][17] It later appeared in 1891 in a case report by Arnold Pick which argued that hebephrenia should be regarded as a form of dementia praecox. Kraepelin first used the term in 1893. In 1899 Emil Kraepelin introduced a broad new distinction in the classification of mental disorders between
dementia praecox and mood disorder (termed manic depression and including both unipolar and bipolar depression). Kraepelin believed that dementia praecox was caused by a lifelong, smoldering systemic or "whole body" process of a metabolic nature that would eventually affect the functioning of the brain in a final decisive cascade. Hence, here
believed the entire body—all the organs, glands and peripheral nervous system—was implicated in the natural disease process.[18] Although he used the term synonymously with "mental defect," and "men
such as in Alzheimer's disease, which typically occur later in life.[19] In 1853 Bénédict Morel used the term démence précoce (precocious or early dementia) to describe a group of young patients who were affected by "stupor".[20] It is sometimes argued that this first use of the term signals the medical discovery of schizophrenia. However, Morel used the term démence précoce (precocious or early dementia) to describe a group of young patients who were affected by "stupor".[20] It is sometimes argued that this first use of the term signals the medical discovery of schizophrenia.
employed the phrase in a purely descriptive sense and he did not intend to delineate a new diagnostic category. Moreover, his traditional conception of dementia differed significantly from that employed in the latter development of
the dementia praecox concept by either Arnold Pick or Emil Kraepelin.[5] Kraepelin's classification slowly gained acceptance. There were objections to the use of the term "dementia" despite cases of recovery, and some defence of diagnoses it replaced such as adolescent insanity.[21] The concept of adolescent insanity or developmental insanity had
been advanced by Scottish psychiatrist Sir Thomas Clouston in 1873, describing a psychotic condition which generally affected those aged 18-24 years, particularly males, and in 30% of cases proceeded to 'a secondary dementia'.[22] Scratch-drawings on the wall in St. Elizabeths Hospital made by a prisoner with "a disturbed case of dementia
praecox". Paul Eugen Bleuler first used the term "schizophrenia translates as "split mind" from the Greek roots schizein (σχίζειν, "to split") and phrēn, phren- (φρήν, φρεν-, "mind").[24] Bleuler coined the term to more
aptly describe the separation of function between personality, thinking, memory, and perception in his patients.[25] Bleuler later published his treatise on the subject, Dementia Praecox oder Gruppe der Schizophrenien, in 1911,[26][27] which is recognised as his magnum opus. Bleuler's treatise describes the fundamental symptoms of the disorder as
being of four A's:[28] flattened Affect, Autism, impaired Association of ideas and Ambivalence.[25] Bleuler sought to differentiate schizophrenia as not a form of dementia, but an entirely separate disorder since his subjects did not suffer from loss or distortion of their memories.[29][28] Bleuler wrote in 1911 of his terminology:[23] I call dementia
precox schizophrenia because, as I hope to show, the splitting of the different psychic functions is one of its most important features. In each case there is a more or less clear splitting of the psychological functions: as the disease becomes distinct, the personality loses its unity. From the creation of the new term, at least two schools of thought arose
after the acceptance of the idea. Some considered Bleuler as having a lesser position of influence with the creation of a novel reality, and instead continued his own thoughts from the initial tradition of Kraeplin, that is, Bleuler inherited the idea, which he then developed. Others, finding Bleuler the greater of the two individuals, believed he in fact
discovered the reality of the disorder anew, using Kraeplin's indication of the existence of disorder but that there was no existing indication by Kraeplin of the new concept in the doctor's written observations and thoughts. [30] In the early 20th century, the psychiatrist Kurt Schneider listed the forms of psychotic symptoms that he thought
distinguished schizophrenia from other psychotic disorders. He termed these as first-rank symptoms. They include delusions of being controlled by an external force; the belief that one's thoughts are being broadcast to other people; and hearing hallucinatory
voices that comment on one's thoughts or actions or that have a conversation with other hallucinated voices.[31] Although they have significantly contributed to the current diagnostic criteria, the specificity of first-rank symptoms has been questioned. A review of the diagnostic studies conducted between 1970 and 2005 found that they allow neither
a reconfirmation nor a rejection of Schneider's claims, and suggested that first-rank symptoms should be de-emphasized in future revisions of diagnostic systems. [32] Many people after the 1908 inception of the term did not accept that schizo-, splitting or dissociation was an appropriate description, and the term would later have more significance as
a source of confusion and social stigma than scientific meaning. [29] In popular culture, the term schizophrenia does not involve a person changing among distinct multiple personality. But for contemporary psychiatry, schizophrenia does not involve a person changing among distinct multiple personality.
to Bleuler's own use of the term to mean "split personality" was by psychologist G. Stanley Hall in 1916, and many early 20th-century
psychiatrists and psychologists can also be found using the term in this sense (some reference Jekyll and Hyde) before a later rejection of this usage went into decline when split personality became known as a separate disorder,
first as multiple personality disorder, and later as dissociative identity disorder.[33] Main article: Political abuse of psychiatry In the first half of the 20th century schizophrenia was considered to be a hereditary defect, and affected people were subject to eugenics in many countries. Hundreds of thousands were sterilized, with or without consent—the
majority in Nazi Germany, the United States, and Scandinavian countries.[34][35] Along with other people labeled "mentally unfit", many diagnosed with schizophrenia were murdered in the Nazi "Action T4" program.[36] In 1933 Dr. Ernst Rüdin, who was in-charge of the Genealogical-Demographic Department of the German Institute for Psychiatric
Research in Munich, expressed his interest in schizophrenia and with the help of Franz Kallmann, supported the idea that schizophrenia was a Mendelian inherited disease. Kallmann believed that the disorder was transmitted by a regressive gene.[37] Both Rüdin's and Kallmann's theories coincided with the growing interest in the idea of
 Rassenhygiene or "race hygiene". The eugenics movement had gained great strength in the United States and Britain. Following suit, in 1933 Rüdin became a guiding force in the prevention of progeny with hereditary defects" which would target individuals with an
intellectual disability, schizophrenia, manic-depressive disorder, epilepsy, Huntington chorea, hereditary blindness and deafness, hereditary alcoholism or "grave bodily malformation." It is suggested by the limited data available that of the 400,000 (1% of the entire population) that were sterilized, 132 000 were sterilized for schizophrenia.[38]
According to E. Fuller Toddy and Robert H. Yolken, it was in 1939 that Hitler asked his private physician and his officials to draft a law that would allow the systematic killing of individuals with mental disorders, sticking to a claim that he had made shortly after assuming office in 1933: "it is right that the worthless lives of such creatures should be
ended, and that this would result in certain savings in terms of hospitals, doctors and nursing staff." In 1932 Berthold Kihn had estimated that mentally ill patients were costing Germany 150 million Reichsmarks per year.[38] In October 1939, German psychiatric hospitals were asked to carry out a survey which established that 70,000 patients would
qualify for. The program was known internally as Aktion (action) T-4. The patients were killed with the use of carbon monoxide which they were given in a closed "shower" room. According to Friedlander, the "overriding criterion" for selection for death in the T-4 program "was the ability to do productive work" [39] useful by doing work such as
dentistry or by pretending to be "asylum director". Psychiatric asylums implemented two diets: minimum calories for those who could not.[citation needed] In the Soviet Union the diagnosis of schizophrenia has also been used for political purposes. The prominent Soviet psychiatrist
 Andrei Snezhnevsky created and promoted an additional sub-classification of sluggishly progressing schizophrenia. This diagnosis was used to discredit and expeditiously imprison political dissidents while dispensing with a potentially embarrassing trial.[40] The practice was exposed to Westerners by a number of Soviet dissidents, and in 1977 the
World Psychiatric Association condemned the Soviet practice at the Sixth World Congress of Psychiatry (41] Rather than defending his claim that a latent form of schizophrenia caused dissidents to oppose the regime, Snezhnevsky broke all contact with the West in 1980 by resigning his honorary positions abroad (42] Harry Stack Sullivan applied the
approaches of Interpersonal psychotherapy to treating schizophrenia in the 1920s viewing early schizophrenia as a problem-solving attempt to integrate life experience than before. [43]: 76 In the early 1930s insulin coma therapy was trialed to treat
schizophrenia, [44] but faded out of use in the 1960s following the advent of antipsychotics. [citation needed] The use of electricity to induce seizures was developed, and in use as electroconvulsive therapy (ECT) by 1938. [45] Frontal lobotomies, a form of psychosurgery, were carried out from the 1930s until the 1970s in the United States, and until
the 1980s in France, involving either the removal of brain tissue from different regions or the severing of pathways, [46] widely recognized as a grave human rights abuse. [45] Antipsychotics were introduced to US hospitals in the 1950s, following the discovery of chlorpromazine in 1952 and
its trialing in French hospitals. Adoption was encouraged by advertising by the Smith, Kline & French company after it received permission to advertise use of the drug within 8 months. In the first report on chloropromazine's use in the US, John
Vernon Kinross-Wright suggested that the drug could be used as an adjunct to psychotherapy to improve its effectiveness. [48]: 33-35 By the 1960s adverts started to imply that antipsychotics explicitly addressed the causes of psychosis using terms like "psychocorrective." The 1973 text book, "The Companion to Psychiatric Studies" asserted that
antipsychotics 'a specific therapeutic effect in schizophrenia, and that the term "tranquiliser" is a misnomer' using the dopamine hypothesis and by 1975 adverts asserted that drugs had an antipsychotic action through acting on dopamine receptors. [48]: 54-55 Anti-psychiatry refers to a diverse collection of
thoughts and thinkers that challenge the medical concept of schizophrenia as a labeling of deviance. Anti-psychiatry represented dissension of psychiatrists themselves about the understanding of schizophrenia in their own field. [49] Prominent
psychiatrists in this movement include R. D. Laing, David Cooper. Related criticisms of psychiatry were launched by philosophers such as Michel Foucault, Jacques Lacan, Gilles Deleuze, Thomas Szasz, and Félix Guattari.[50] Anti-psychiatrists agree that 'schizophrenia' represents a problem, and that many human beings have problems living in
modern society.[citation needed] But they protest the notion that schizophrenia appear crazy because they are intelligent and sensitive beings confronted with a mad world. The sane patient can choose to go against medical advice, but the
insane usually cannot. Anti-psychiatry often describes the institutional world as itself pathological and insane because of the way it subordinates human beings to bureaucracy, protocol, and labels. [49] In his book, The Divided Self, published in 1960, R. D. Laing proposed a psychodynamic model of schizophrenia using the concept of ontological
security. He presented a model where schizophrenia is the attempt of the "self", the attention of the mind, to escape the experiences of the world, the "body". The understanding and connection of others, he argued, is felt as either an attack or "smothering understanding" while simultaneously being longed for. Laing posited that in this state the "self"
could become angry, hateful, and split and that the strange language of metaphor present in schizophrenia was simultaneously an attempt to avoid being understood, and to be partially understood that true
understanding of the self can resolve schizophrenia.[51]:137 In 1970 psychiatrists Robins and Guze introduced new criteria for deciding on the validity of a diagnostic category[52] and proposed that cases of schizophrenia but a separate condition.[53] In the early 1970s, the diagnostic
criteria for schizophrenia was the subject of a number of controversies which eventually led to the operational criteria used today. It became clear after the 1971 US-UK Diagnostic Study that schizophrenia was diagnosed to a far greater extent in America than in Europe. [54] This was partly due to looser diagnostic criteria in the US, which used the
DSM-II manual, contrasting with Europe and its ICD-9. David Rosenhan's 1972 study, published in the journal Science under the title "On being sane in insane places", concluded that the diagnosis of schizophrenia in the US was often subjective and unreliable. [55] The 1970s controversies led to the revision not only of the diagnosis of schizophrenia,
but the revision of the whole DSM manual, resulting in the publication of the DSM-III in 1980.[56] The revision was based on Feighner Criteria and Research Diagnostic Criteria that had in turn developed from Robins's and Guze's criteria, and which were intended to make diagnostic make diagnostic Criteria and Research Diagnostic 
criteria for schizophrenia have been proposed and evaluated.[57] The DSM-IV of 1994 showed an increased focus on an evidence-based medical model, with the diagnostic criteria for schizophrenia slightly adjusted to require one month of positive symptoms instead of one week.[58] In 2002 in Japan the name was changed to integration disorder (統合
失調症), and in 2012 in South Korea, the name was changed to attunement disorder (思覺失調), to refer to psychosis since 2001, [63] keeping the original "split-psyche" name of schizophrenia. [64] Subtypes of
schizophrenia are no longer recognized as separate conditions from schizophrenia by DSM-5[65] or ICD-11.[66] Before 2013, the subtypes of schizophrenia were classified as paranoid, disorganized, catatonic, undifferentiated, and residual type.[67] The subtypes of schizophrenia were eliminated because of a lack of clear distinction among the
subtypes and low validity of classification. [66][68] Psychiatry portal Montreal experiments Physical health in schizophrenia Schizophrenia Schizophrenia Became a Black Disease Catastrophic schizophrenia Protest Psychosis: How Schizophrenia Became a Black Disease Catastrophic schizophrenia Protest Psychosis: How Schizophrenia Became a Black Disease Catastrophic schizophrenia Protest Psychosis: How Schizophrenia Protest Psychosis: How Schizophrenia Became a Black Disease Catastrophic schizophrenia Protest Psychosis: How Schizophrenia Psychosis: How
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