

Finding Common Ground Among Human Cultures

Every one of us is unique. Curiously enough, we are so unique it is difficult to determine what we have in common. Scientists have found it



▲ **Figure 1.** Cave painting depicting a San Bushmen hunting party about to attack a herd of elephants. San Bushmen were one of the hunter-gatherer societies analyzed in the study.

surprisingly hard to find which social traits are truly “human,” and not the result of local cultural evolution within particular regions of the planet. In a study recently published in *Science*, a group led by Dr. Kim Hill and Prof. Robert Walker (from Arizona State University and the University of Missouri, respectively) sought out to discover the ancestral biological characteristics of our species history, which eventually favored the acquisition of culture (1).

In collaboration with anthropologists from across the US, the authors obtained demographic data from 32 present-day hunter-gatherer societies around the world. Members of these societies

likely live similarly to how all humans lived before developing large-scale cultural systems (1). The authors focused on co-residence patterns and asked questions like: What is the mean number of adult individuals who are closely related and live together? Or how likely are siblings of the opposite sex to live together?

The findings suggest that cooperation in these societies occurs via extensive networks of social interactions between unrelated individuals. Adult sisters and brothers tend to live together, but the majority of individuals that co-reside in a tribe are usually unrelated (1). This is a rare trait among primates, and points to large-scale non-kin sociability as a possible explanation for why humans eventually evolved complex cultures (1). A primitive “good neighbor” policy may indeed be the reason why societies arose, in contrast to the archaic view of foraging cultures as purely patriarchic kin-based groups. The data was collected from present-day populations and is thus only a proxy for ancestral behaviors, which are hard to infer from archeological remains. Nevertheless, the evidence it provides in favor of non-kin cooperation is strong, and calls for new mathematical models to explain the origin and evolution of human culture. **H**

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A Second Alarm System

Second half of spring semester, in the last sprint towards completing theses, final papers, and final exams, us college students unfortunately prioritize sleep as second-tier. In our rush to complete writing assignments the day before the due date, we allow ourselves a few hours, or even just a few minutes, of blissful sleep before we again slave away at our work. We program our phones, and then our alarm clocks...and then ask roommates to wake us up at a certain time. As shown by our desperate measures in ensuring that we wake up, there comes reassuring news that our own biological system may also use a multi-tiered approach

to detect environmental cues and signal us to wake (1).

Drosophila express a light-sensitive protein, CRYPTOCHROME (CRY), that helps reset the animal’s circadian clock each day (2). It was found that flies with a CRY mutant show less response to changes in light schedule which would normally cause change in behavioral cycles (2)—indicating the important nature of the protein in regulating circadian cycle. CRY interacts with TIMELESS (TIM), a circadian protein, and promotes TIM’s interaction with the protein JETLAG (3,4). This cycle results in the

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degradation of TIM, which resets the circadian clock (3,4). In addition to this cycle, CRY was also found to affect specific circadian pacemaker neurons, called large lateral ventral neurons (1). These neurons control daily rhythmic behavior and a select eight of the neurons explicitly work as light detectors (5). Different light level exposure controlled the rate of action potential generation of those neurons (1); the level of light produced during midmorning correlated with the greatest increase in rate of neuron firing (1). This process is dependent on CRY (1). As the process was also TIM-independent, the results suggest that CRY is involved in two distinct mechanisms to detect light and determine behavioral cycle (1).

The study shows a possible parallel function in mammals, as the photosensitive retinal ganglion

cells express similar patterns of response to light (increase in action potential generation) (6)—reassuring us sleepless students that even if we don't hear the alarm go off, our own neurological wiring will wake us up, at one time or another. ■

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Large Hadron Collider: A Glimpse of the Early Universe

Ever since it was first tested at the end of 2009, the Large Hadron Collider (LHC) attracted a great deal of public and media attention. Built at a cost of more than \$9 billion dollars, the LHC has the potential to answer fundamental questions in theoretical physics and expand our understanding of the universe. The LHC, like all accelerators, propels subatomic particles at very high energies and observes the even smaller particles released from these collisions. The LHC will eventually force particle beams with energies of 7 trillion electron volts into head on collisions millions of times every second (1). Particles travel through vacuum tubes wrapped by super cooled electromagnets, which accelerate the particles to nearly the speed of light before they finally collide inside a detector. The properties and trajectories of released particles are studied to identify the characteristics of the particles produced. One of the major goals of the project is to find the elusive Higgs boson, a particle that is predicted by the standard model of physics to give mass to all other particles. The LHC may also answer more esoteric questions about whether the extra spatial dimensions predicted by string theory actually exist, and the composition

of “dark matter,” a substance that makes up 25% of the universe compared to the 5% made from our “ordinary” matter (1). Last month, researchers at Syracuse University reported observing a new type of decay of a particle called the B meson, which contains both a quark and an antimatter anti-quark. B mesons would have existed in the early universe, and this discovery may provide some clues about why the universe contains large amounts of matter and comparatively little antimatter (2). The LHC gives physicists a glimpse into what the universe may have looked like immediately following the big bang, thereby providing some clues about how the structure of the universe evolved from that time to its current form (1, 2). ■

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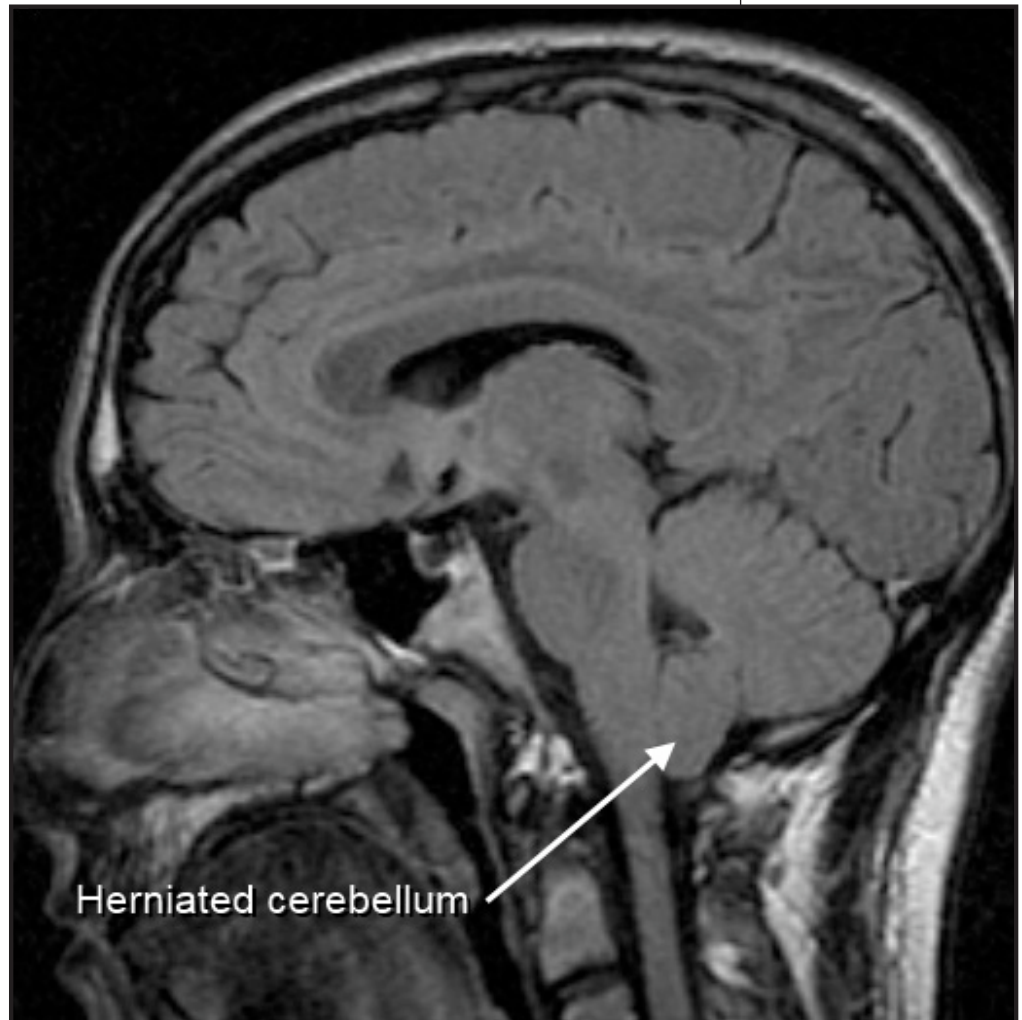
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Chiari Malformation

By Anirudh Penumaka

Neurosurgery as a distinct discipline only arose in the first few decades of the 20th century, and the inherent difficulty of these surgeries was compounded by a lack of understanding about the brain as well as the absence of the advanced imaging techniques used today. Famous surgeons like Harvey Cushing, Gazi Yasargil, and Wilder Penfield would later advance neurosurgery by developing methods to diagnose and remove brain tumors, treat aneurysms, map the motor cortices, and surgically treat epilepsy. While brain tumors are the most well-known conditions requiring neurosurgery, there are several types of disorders which have severe symptoms due to changes to the complex architecture of the brain. First characterized by Hans Chiari in 1891, Chiari malformation (CM) is a rare neurological condition that affects parts of the brain located near the base of the skull. CM most often occurs due to a congenital malformation, which can be detected soon after birth in severe cases, but is usually not symptomatic in less severe cases until adulthood. Chiari malformation occurs when tissue at the base of the cerebellum (tonsils) herniates through an opening called the foramen magnum into the spinal canal (1). The displacement of the cerebellar tonsils is a result of compression due to the reduced size of the posterior fossa, a region of the brain which normally contains the cerebellum, medulla, and pons. The cerebellum, which is in charge of balance and motor control, faces increased pressure from the posterior fossa, which forces the lower portions of the cerebellum downward (2).

Chiari malformations are described according to their degree of severity with type I cases being the least acute. Type I CM occurs when the herniated tonsils partially or completely occlude

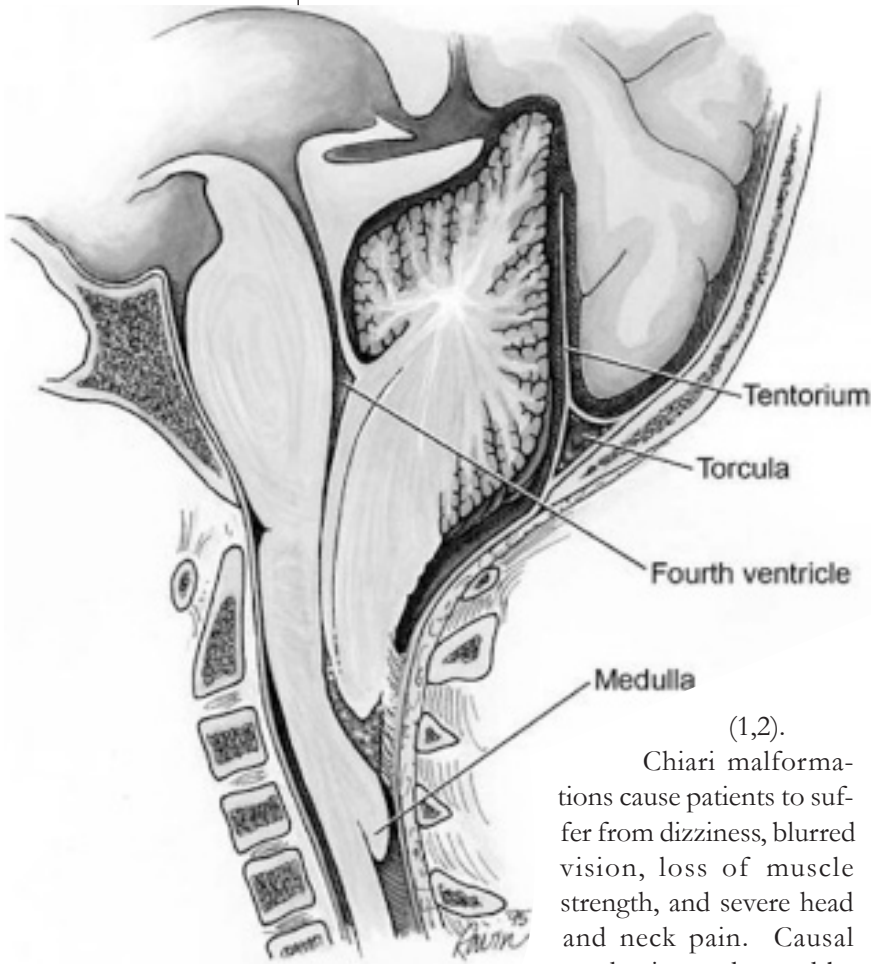


the “pulsatile flow of cerebrospinal fluid (CSF)” between the intracranial space and the spinal canal (1). Syringomyelia, frequently associated with CM1, is the formation of a cavity inside the spinal canal secondary to the blockage of normal CSF flow. The cavity, called a syrinx, can damage nerves as it grows inside the spinal cord to cause a loss of sensation, paralysis, and scoliosis in some cases. In type II CM, the fourth ventricle, medulla, and cerebellum all descend into the spinal canal and the spinal cord fails to

▲ Figure 1. MRI of Type I Chiari Malformation

credit: http://commons.wikimedia.org/wiki/File:MRI_of_human_brain_with_type_1_Arnold-Chiari_malformation_and_herniated_cerebellum.jpg

scoliosis in some cases. In type II CM, the fourth ventricle, medulla, and cerebellum all descend into the spinal canal and the spinal cord fails to close completely (Myelomeningocele). Finally type III and type IV CM are fairly uncommon and produce conditions that have high mortality rates or major complications. In particular, type IV CM may involve abnormal morphology of the cerebellum and other structures of the brainstem



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Figure 2. Artist's representation of a Chiari II malformation showing the points of potential obstruction

(1,2). Chiari malformations cause patients to suffer from dizziness, blurred vision, loss of muscle strength, and severe head and neck pain. Causal mechanisms advanced by Gardner, then modified by Williams, suggest that pressure gradients between the cranial and spinal compartments of CSF cause herniation of the cerebellum as CSF is inhibited from reentering the spinal compartment. It is the consequences of this pressure gradient of cerebrospinal fluid (CSF) at the base of the brain that manifests as the observed neurological symptoms (3,4). Treatment for the symptoms of CM and to prevent progression of the disorder requires surgical intervention. One method of treatment, called posterior fossa decompression, involves remov-

ing a small piece of bone at the base of the skull in addition to the uppermost cervical vertebra of the spine. The removal of bone reduces pressure on the cerebellum, and expansion of the dural covering of the brain with a patch constructs additional space around the site of obstruction. This procedure creates space around the blockage in order to re-establish free flow of CSF. While not always done, the tonsils of the cerebellum may also be removed to decrease the size of the blockage at the craniovertebral junction (1). One issue that is still debated is the method used to expand and seal the dura. In the pericranial autograft method, a piece of the layer lining the outside of the skull, called the pericranium, is grafted to the incision site to close the dura. Other methods of dural patching involve synthetic dural substitutes and xenogenic or cadaveric grafts. While various surgeons have reported different results, the pericranial autograft method avoids issues of immune rejection and shows a great deal of promise (5, 6).

Based on detailed images provided by MRI studies, it is estimated that Chiari malformation occurs in approximately 0.1% to 0.5% of the population, much greater than imagined before the widespread use of MRI. While treatment for Chiari Malformation has existed for a long time, research to improve current methods will continue to generate advances and innovations.

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