A step-by-step process that explains replication (cell division) is outlined in the documents affixed to this article.
They provide a verifiable explanation of how epigenetic activities could and should be monitored to prevent chronic diseases.

How does a cell maintain its identity during replication?

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Prior to cell division, chromosomes are seemingly a jumbled mess. During cell division, parent cell chromosomes and their duplicates sort themselves out by condensing, becoming thousands of times more compact than at any other time. Researchers have long assumed that genes become "silent" during cell division, not being transcribed into proteins or regulatory molecules. This has left open the question of how genes get properly re-activated after cell division. Now, researchers in the Perelman School of Medicine at the University Pennsylvania have found that gene expression actually continues during cell replication. Their findings are published this week in *Science*.
"We looked at this question from the point of view of answering what controls cell identity and how can we harness that for cell reprogramming - for instance, to stop cancerous replication or engineer a cell to steer the direction of its 'personality,' so to speak," said senior author Kenneth S. Zaret, PhD, director of the Penn Institute for Regenerative Medicine and the Joseph Leidy Professor of Cell and Developmental Biology. "The set of genes a cell expresses determines if it's a skin cell, nerve cell or a heart muscle cell, among the 200 or so different cell types found in the human body."
Past studies in this area also sought to pinpoint the best time to intervene in order to change a cell's fate. The current *Science* paper shifts to exploring how a cell goes from a quiet gene state to a fully active staff of genes and regulatory molecules that control the outcome of the entire cell's identity.

First author Katherine C. Palozola, a doctoral candidate in the Zaret lab, is the first to find a way to look at gene activity in a living cell during division. Using a human liver cell line, she labeled the nucleic acid uridine (one of the four gene messenger building blocks) and followed it to see which genes were still active during replication.
"We were surprised that gene expression was still on - albeit at a low level - during replication," Palozola said.

Although chromosomes are extremely compact during cell division, with sequences for regulatory molecules buried and previously presumed to be unavailable to be transcribed, Palozola found that most genes and their nearby regions that promote gene function are still actively expressed. She discovered how cells wake up after cell division and recall "who they are." What ultimately drives cell differentiation are sequences of enhancer molecules located away from the gene they act on.

The laboratory of Gerd Blobel at the Children's Hospital of Philadelphia had previously shown that these far-away modifiers "nap" during division, since it only lasts about 30 minutes - relatively quickly in biological terms - and come back online after a cell division cycle is complete.

"The most amazing thing about this study is that in the end, we had to throw what we thought we knew about this basic aspect of gene regulation out the window," Zaret said. "The findings indicate that we need to think about how promoters, rather than enhancers, are regulated during cell division. This refocusing will tell us how a cell's identity, as defined by the genes it expresses, is retained through cell division. We hope it will improve our ability to deliberately change a cell's identity to create new cells and tissues for therapeutics and research."

Explore further: Impaired DNA replication can cause epigenetic changes inherited for several generations
More information: Mitotic transcription and waves of gene reactivation during mitotic exit, *Science* (2017).**science.sciencemag.org/lookup/ … 1126/science.aal4671**

Journal reference: Science

Read more at: https://phys.org/news/2017-09-cell-identity-replication.html#jCp

The following are crucial issues to consider for how and why cells must be “healthy” in prevent chronic diseases that can result from problems associated with replication (i.e. division).

These step-by-step explanations are provided to overcome terminology problems that can occur when discussing translations between particle physics and biomedical research applications.

The following is provided to heighten awareness to terminology issues.

http://www.mcfip.net/upload/Jargon%20Problem%20in%20Health%20and%20Science.pdf

Disruption of mechanisms that regulate division and reassembly based on the principles of particle physics and quantum mechanics (i.e. self-assembly into entanglement into “genes that encompass activities that

regulate DNA repair) can be complex and confusing unless the information is provided in a linear flow and supported by facts. The numerous hyperlinks in this document are provided as a means of minimizing confusion and to enable interested parties to independently verify assertions.

The size of molecules (scale) must be considered because activities within organelles within cells will be at scales far below nano; i.e. pico to femto or femto to attoscale. Accordingly, current technology does not allow for visualization of the interactions between elements that constitute epigenetic signaling. To overcome this obstacle, having identified the elemental constituents of epigenetic signaling molecules from retrospective review of thousands of studies, we have been able to translate signaling that is identified through research on organelles through our knowledge of the same signaling molecules that are cell-surface. Our modeling for cell-surface (small molecule activities) is described below:

http://www.mcfip.net/upload/Cell%20Surface%20Signaling%20Molecule%20Formation%207-2017.pdf

Numerous studies have identified calcitonin as the signaling mechanism for cell division. Using our modeling for elemental constituents, calcitonin was matched to be the three forms of vitamin D.

Activation of vitamin D was identified as the mechanism created by the trefoil of Abl1 - Abl2 and BCR-Abl that is explained in the following hyperlink that describes DNA repair and anabolic activity that forms entanglement of "genes." Having the aromatic amino acids as constituents that are activated by UVB light waves at 256 - 280 nm, **keeping scale in mind**, with near certainty, arching from electrolytes provides intracellular light that is within that range.

Note: Explanation for light waves and cellular changes is addressed here for discussion purposes.

http://www.mcfip.net/upload/Optogenetics%20-%20Confirmation%20of%20Value.pdf

Translation of optogenetics into photosynthesis for cells other than plants can be discussed using our verifiable model for photopharmacology.

Cellular health must be optimal to ensure division and to prevent copy error mutations.

The document affixed to this one explains the mechanisms of nuclear restorer signaling that regulates a key facet of cell division; i.e. to prevent formation of a third chromosome that can lead to cancers and other chronic diseases.

A second level of DNA repair (other than NRF signaling) is the one that forms entanglement that encompasses anabolic and catabolic activities; i.e. binding and reassembly post-division of cells as well as the ability to disassemble the contents of cells (catabolic activity) to allow for division to occur. Those activities are outlined below for discussion purposes.

http://www.mcfip.net/upload/Epigenetics%20-%20DNA%20Repair%20-%20Copy%20Errors.pdf

Note: It is disruption of anabolic and catabolic mechanisms that created copy errors as cells divide with up to 65% of all chronic diseases be linked to the problem.

Our modeling of NRF activity is likely to account for one half of the remaining 1/3 of the causes of chronic diseases.

The following is provided for discussion purposes to explain why the instances of chronic diseases are increasing.

http://www.mcfip.net/upload/Copy%20Errors%20-%20Epigenetic%20Inheritance.pdf

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DNA Repair: Nuclear Restorer Factors (NRFs)

Our modeling process, as supported by existing peer-reviewed studies, identified the nuclear restorer factors as the DNA repair mechanism that regulates signaling between the two chromosomes that exist in normal (healthy) cells; one in the nucleus and one in the mitochondria.

Relative to Nuclear restorer factors (NRFs), the following appeared in an article from 2013: http://medicalxpress.com/news/2013-08-witness-cell-division-cancer.html

“Roughly 14 percent of breast cancers and 35 percent of pancreatic cancers have extra sets of chromosomes, compared to normal cells that have two.” **“Roughly 14 percent of breast cancers and 35 percent of pancreatic cancers have extra sets of chromosomes, compared to normal cells that have two.”**

It should be noted that our modeling of NRF activities identifies these signaling molecules as being regulated by the interactions of gasotransmitters and oxygen. With near certainty, imbalances such as one between nitric oxide and hydrogen sulfide will disrupt NRF activity and create cell division problems that can result in the formation of a third set of chromosomes.

The activities of nuclear restorer factors that are part of DNA repair are described here,

http://www.mcfip.net/upload/Interaction%20between%20Chromosomes%20xx.pdf