

Career Choices

Academics

Research

Administration

Agnes A. Day, Ph. D.

Chairman

Department of Microbiology

College of Medicine

Howard University

Washington, DC 20059

First Science Experiment

Experiment # 1



Materials

Can of Baking Soda
Glass of Water



Methods

Add water to baking soda in
can
Replace cap
Shake



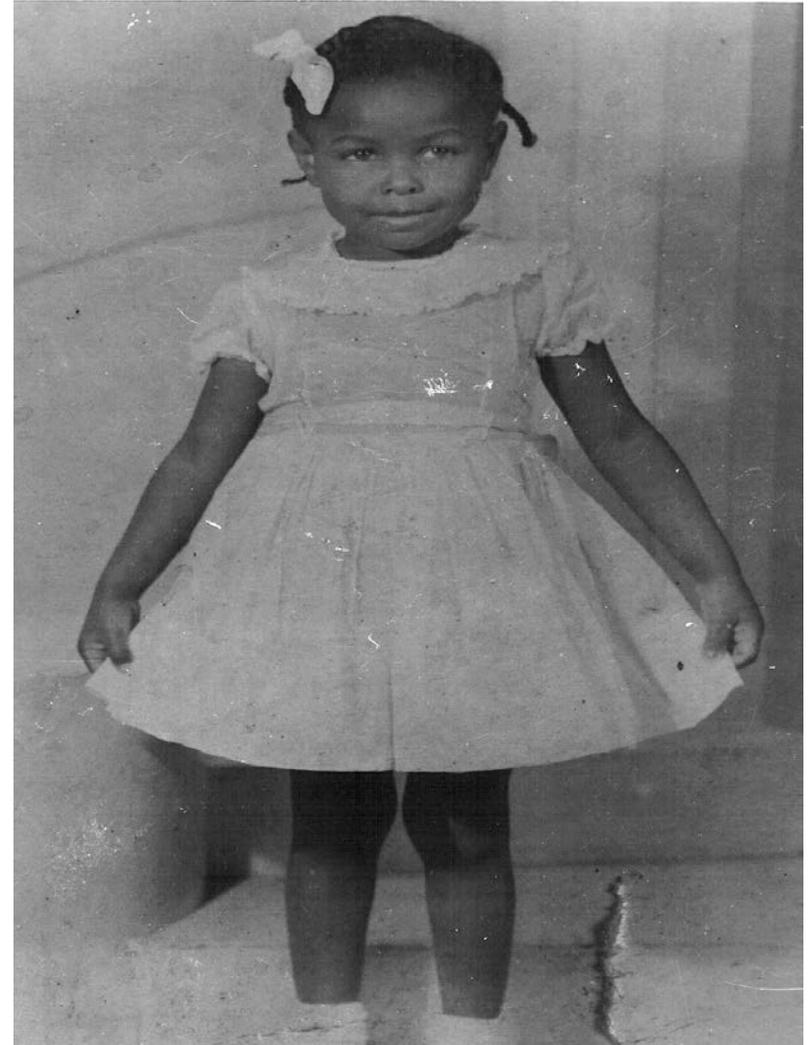
Results

Fizzy explosion 😊!
messy kitchen 😞 !!
spanking 😞 😞 !!!



Conclusions

1. The spanking was worth it
2. Conduct next experiment
when no one is home.



High School Years

- 11th grade summer research at U. FL “Sleep Deprivation: Impact on Short-term Memory and Long-term Retention”.



*Miss
Mainland*



Agnes Lasiter

The College Years

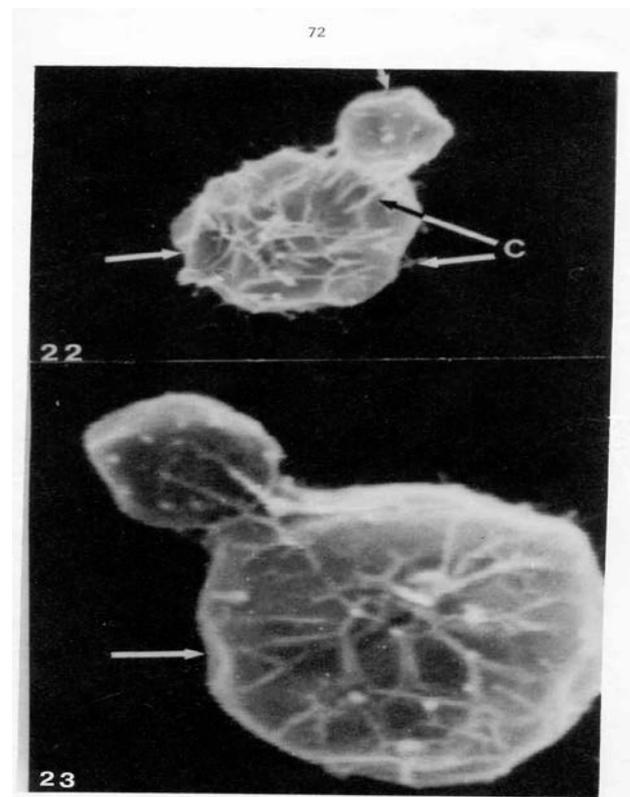
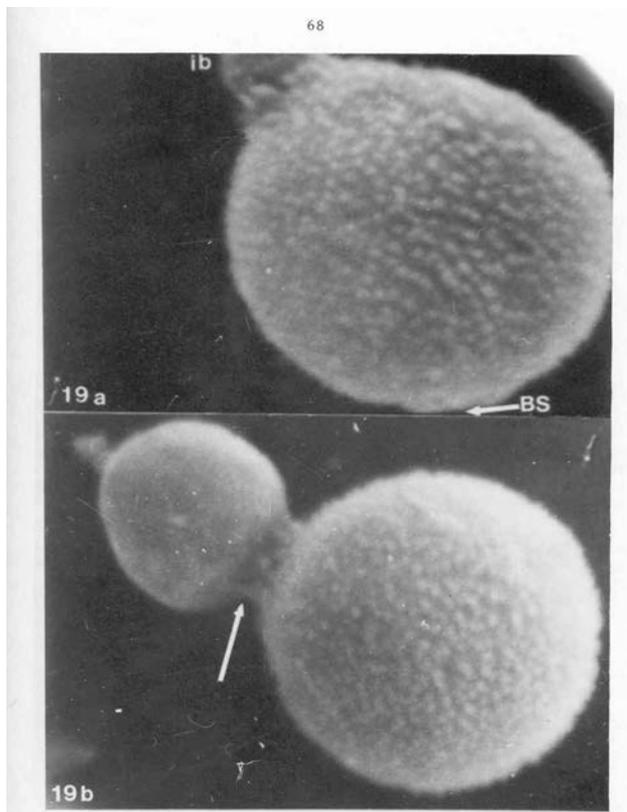
Bethune-Cookman College
Daytona Beach Florida

- **Biology Pre-Med Major**
 - Research: Impact of Cape Canaveral Space Center on Regional River Estuaries
- **Summer Pre-Med Program**

Fisk University / Meharry Medical College
- **Audit of Bacteriology**
 - Dr. Gregory Marlins

Graduate Research

Cryptococcus neoformans



Hard Work Pays Off !



Post Doctoral Experiences

Bone Research Branch, NIDR, NIH



Publications

Volume 14 Number 11 1986

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Calcium regulation and bone metabolism: Basic and clinical aspects, vol. 9
D.V. Cohn, T.J. Martin, P.J. Meunier, editors

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CHARACTERIZATION OF PG(II) CORE PROTEIN AND THE EXPRESSION OF PG(II) mRNA IN BONE AND SKIN CELLS

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LARRY W. FISHER AND JOHN D. TERMINE

Bone Research Branch, National Institute of Dental Research, National
Institutes of Health, Bethesda, Maryland 20892 (USA)

INTRODUCTION

Proteoglycan II (PgII, $m_r \approx 80,000-120,000$) is the major proteoglycan in bone and appears localized within the mineralizing bone matrix (1). Pg (II) has been shown to affect both the rate of formation and final size of Type I collagen fibers (2,3) and has been implicated as binding specifically to gap regions of collagen fibrils in many connective tissues (4). While similar Pg(II) molecules have been described in articular cartilage (5) tendon (6) skin (7) and sclera (8), the precise structural relationship of these proteins to each other has not definitively been elucidated. In this report we describe the immunological relationship of the Pg(II) core proteins of bone to that of tendon, skin, and cartilage and, further, using cloned Pg(II) cDNA have determined the relative concentration of Pg(II) mRNA in bone and skin compared to another matrix protein, osteonectin.

MATERIALS AND METHODS

RNA Extraction and Cell Free Translation. RNA was extracted from cultured fetal bovine bone (9), skin, and adult tendon as described (10). Total RNA was quantitated by its optical density at $\lambda 260$ and 1.0 μg translated in a rabbit reticulocyte lysate system (New England Nuclear) using ^{35}S methionine as tracer. Radioactive proteins were immunoprecipitated as described (11) and analyzed after electrophoresis in 10% acrylamide gels by fluorography.

Northern Analysis. A 3.5 μg aliquot of total RNA was electrophoresed in 1.2% formaldehyde agarose gels and transferred to nitrocellulose as described (12). Insert DNA from an osteonectin cDNA (11) and a Pg(II) cDNA (13) were radiolabeled by nick-translation (Amersham) and hybridized to filter bound RNA. Hybridization was carried out in 50% formamide (11) at 37° and filters washed and autoradiographed as described (11).

Osteonectin mRNA: distribution in normal and transformed cells

Marian F. Young, Mark E. Bolander*, Agnes A. Day, Camille I. Ramis, Pamela Gehron Robey, Yoshihiko Yamada* and John D. Termine

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Received 6 March 1986; Accepted 7 May 1986

ABSTRACT

Overlapping cDNA clones encoding bovine osteonectin were isolated from a λ gt11 expression library constructed from bovine bone cell mRNA. The longest clone, λ On 17 (insert size 2.0 kb) was studied in detail. The clone was shown to encode osteonectin by hybrid select translation experiments and by DNA sequence analysis. Northern analysis of bone cell RNA showed the length of the osteonectin mRNA to be 2.0 kb. Osteonectin message was found in bone but not in soft tissue (liver and brain) preparations consistent with the distribution of the protein in these tissues. On the other hand, osteonectin message was observed in tendon, a tissue in which little or no osteonectin protein is found *in vivo*. Hybridization of osteonectin cDNA was detected in cells from a number of species including human, rat, mouse and chick. The level of osteonectin mRNA was drastically decreased in chick embryo fibroblasts transformed by Rous sarcoma virus.

INTRODUCTION

Osteonectin is a major non-collagenous protein of bone. It is a phosphorylated glycoprotein with an apparent molecular weight of 38,000 on SDS gels (1). While Type I collagen is the major protein component of the bone matrix (>90% of protein content), it is widely distributed in non-mineralizing connective tissues such as skin, vasculature and tendon (2). Osteonectin, on the other hand, is localized predominantly to the osteogenic compartments of bone (3) and is an avid binding protein for calcium, hydroxyapatite and collagen (3,4). It also promotes the deposition of calcium phosphate mineral onto type I collagen *in vitro* (3). Thus it has been proposed that osteonectin may play a role in structural bone mineralization *in vivo* (3).

The regulation of osteonectin expression appears to be complex. Cell free translation of osteonectin mRNA produces a protein approximately 6,000-9,000 M_r larger than that extracted from intact tissue (5,6) suggesting that the secreted protein is extensively modified. In addition, despite observations that this protein is unique to bone tissue,

Back to My Beloved Alma Mater

JOURNAL OF CELLULAR PHYSIOLOGY 156:497-514 (1993)

Expression of Transforming Growth Factor α Antisense mRNA Inhibits the Estrogen-Induced Production of TGF α and Estrogen-Induced Proliferation of Estrogen-Responsive Human Breast Cancer Cells

N.J. KENNEY, T. SAEKI, M. GOTTARDIS, N. KIM, P. GARCIA-MORALES, M.B. MARTIN, N. NORMANNO, F. CIARDIELLO, A. DAY, M.L. CUTLER, AND D.S. SALOMON*

Department of Microbiology, College of Medicine, Howard University, Washington, DC 20001 (N.J.K., A.D.), Tumor Growth Factor Section, Laboratory of Tumor Immunology and Biology, Division of Cancer Biology, Diagnosis, and Centers, National Cancer Institute, Bethesda, Maryland 20892 (N.J.K., N.N., N.K., M.L.C., D.S.S.); Department of Surgery, Research Institute for Nuclear Medicine and Biology, Hiroshima University, 1-2-3 Kasumi, Minami-ku, Hiroshima 734, Japan (T.S.); Vincent Lombardi Cancer Research Center, Georgetown University Hospital, Washington, DC 20007 (M.G., P.G.-M., M.B.M.); Cattedra di Oncologia Medica, II Facolta' degli Studi di Napoli, Via S. Pansini 5, 80131 Napoli, Italy (F.C.)

To ascertain if 17 β -estradiol (E2)-induced proliferation could be attenuated by blocking the expression of endogenous transforming growth factor α (TGF α), estrogen receptor (ER)-positive, estrogen-responsive MCF-7 or ZR-75-1 cells and ER-negative, estrogen-nonresponsive MDA-MB-468 or HS-578T cells were infected with a recombinant amphotropic, replication-defective retroviral expression vector containing a 435 base pair (bp) *Apa1-Eco R1* coding fragment of the human TGF α cDNA oriented in the 3' to 5' direction and under the transcriptional control of an internal heavy metal-inducible mouse metallothionein (MT-1) promoter and containing the neomycin (*neo*) resistance gene. E2-stimulated expression of endogenous TGF α mRNA was inhibited by 4–5-fold, and the production of TGF α protein was inhibited by 50–80% when M-1 mass-infected MCF-7 or MZ-1 mass-infected ZR-75-1 cells were treated with 0.75–1 μ M CdCl₂, whereas in comparably treated parental MCF-7 or ZR-75-1 cells there was no significant effect upon these parameters. E2-stimulated anchorage-dependent growth (ADG) and anchorage-independent growth (AIG) of the M-1 or MZ-1 cells was inhibited by 60–90% following CdCl₂ treatment. In contrast, neither the ADG nor AIG of the parental noninfected MCF-7 or ZR-75-1 cells that were maintained in the absence or presence of E2 was affected by comparable concentrations of CdCl₂. The ADG and AIG of TGF α antisense MD-1 mass-infected MDA-MB-468 cells that express high levels of endogenous TGF α mRNA were also inhibited by 1 μ M CdCl₂, whereas the ADG and AIG of MH-1 mass-infected HS-578T cells, a TGF α -negative cell line, were unaffected by CdCl₂ treatment. These results suggest that TGF α may be one important autocrine intermediary in regulating estrogen-induced cell proliferation.

Teaching

Kaiser-Permanente Outstanding Teaching Award
Medical, Dental, Pharmacy, Allied Health and Graduate courses

Mentoring

Over 400 undergrad, and professional students
Research Advisor to 25 Ph. D. students
40 Dissertation Committees
1,000 High School students during summers
2 Post-doctoral Research Associates
50 Undergraduates during summers/ Senior Thesis
ASM William A. Hinton Award for Research Mentoring and Training

Research

Garnered over \$2,500,000 in **individual** research grants
Outstanding Researcher Award

Service

Associate Director for Basic Research, HUCC
Chairman, Department of Microbiology
Committee member of ASM, AACR and AAAS

Achievements

- Teaching
 - Kaiser-Permanente Outstanding Teaching Award
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 - **Chairman, Department of Microbiology**
 - Committee member of ASM, AACR and AAAS

Roadmap to Success in Academia

- Publish, Publish, Publish
- 2yr Plan, 5yr Plan, 10yr Plan
- Collaborations and Interdisciplinary Research
- NIH Career Workshops, AAMC Workshops
- Promotions and Tenure
- “Be Careful What You Wish For...”