

MEDICINE ELSEWHERE

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Chen LT, Gilman AG, Kozasa T. A candidate target for G protein action in brain. J Biol Chem 1999;274:26931-26938.

Heterotrimeric G-proteins play an essential role as transducers of information by coupling many cell surface receptors to effectors at the plasma membrane. They are classified in 4 main subgroups: Gs, Gi, Gq, G12.

Members of the $G_{i\alpha}$ subfamily ($G_{i\alpha}$, $G_{o\alpha}$, $G_{t\alpha}$, $G_{g\alpha}$, $G_{z\alpha}$), particularly $G_{o\alpha}$ and $G_{i\alpha}$, constitute roughly 1% of brain membrane protein; the only effector known so far to be coupled to these two proteins and also to some isoforms of $G_{z\alpha}$, is adenylyl cyclase.

In this study, Chen et al. have elucidated a new effector candidate for G-protein action, named GRIN1 and they have also identified a homolog of that protein using public databases, designated as KIAA0514 (GRIN2). GRIN1, shown to be a novel protein without substantial homology to known protein domains, was homologous to GRIN2 for ~100-150 amino acid residues at the carboxyl terminus which is the $G_{o\alpha}$ -binding domain. Both proteins interacted preferentially with activated members of the Gi subfamily of G-protein α -subunits. GRIN1 was specifically expressed in the brain and found in substantial enrichment in membranes from neuronal growth cones, as $G_{o\alpha}$.

G_o is responsible for receptor-mediated inhibition of voltage sensitive N-type or P/Q-type Ca^{2+} channels in presynaptic nerve terminals, but this effect appears to be mediated by the G-protein $\beta\gamma$ subunit complex. $G_{o\alpha}$ is a weak

inhibitor of some isoforms of adenylyl cyclase, but the physiological significance of this is difficult to evaluate. $G_{o\alpha}$ has also been hypothesized to regulate neurite extension. Binding of $GTP\gamma S$ to $G_{o\alpha}$ is stimulated by GAP43, an abundant growth cone protein that is important for neural pathfinding. The expression of both GAP43 and $G_{o\alpha}$ starts in brain regions when differentiated neurons begin to extend neurites.

In this paper, Chen et al. have shown that GRIN1 and GRIN2 induces extensive outgrowth of neurites from Neuro2a cells when coexpressed with activated forms of $G_{o\alpha}$. The result implies interaction between proteins *in vivo*, although not necessarily a direct one. However, the fact that $G_{o\alpha}$ does interact directly with both GRIN1 and GRIN2 *in vitro* suggests that these proteins may function physiologically as downstream targets for $G_{o\alpha}$ and/or other members of the $G_{i\alpha}$ subfamily to regulate neurite outgrowth.

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Van Keymeulen A, Roger PP, Dumont JE, Dremier S. TSH and cAMP do not signal mitogenesis through Ras activation. Biochem Biophys Res Commun 2000; 273:154-158.

Activation of the small G protein Ras by receptor tyrosine kinases or serpentine receptors is generally considered to be essential for G1 phase progression and mutagenesis. In various systems, Ras function is required throughout G1 phase for D1 expression, p27^{kip1} downregulation, pRb phosphorylation and S phase initiation.

Dog thyroid epithelial cells in primary culture constitute a well-characterized model for

studying cell cycle regulation by TSH and cAMP. In response to TSH, cAMP is generated in these cells; but interestingly, the cAMP-dependent mitogenesis is not associated with p42/ p44 MAP kinases phosphorylation and activation, suggesting that Ras is not involved in activation of this pathway. However, in the thyroid cell line, WRT, the inhibition TSH/cAMP stimulated DNA synthesis with mutants of Ras suggests an intermediary role of Ras in cAMP-dependent mitogenesis.

This study by Van Keymeulen et al. aims to reevaluate the involvement of Ras in cAMP-dependent mutagenesis. The contents of Ras-GTP were assessed in normal dog thyrocytes using an assay based on immunoreactivity between GTP-bound Ras and Raf-RBD. EGF, HGF and TPA, which trigger mitogenesis in dog thyrocytes by cAMP-independent pathway, have previously been shown to activate p42 and p44 MAP kinases in these cells. These agents were shown to lead to strong activation of Ras in this study. Insulin and carbachol which when used alone are not mitogenic factors in thyrocytes but trigger a tyrosine kinase cascade and increase the cell size, were found to activate Ras, but more weakly, than growth factors and TPA. By contrast, the basal level of GTP-Ras was slightly reduced by TSH and forskolin, a direct activator of adenylyl cyclase. In summary, this study demonstrates that TSH and cAMP which induce both proliferation and differentiation expression, do not activate Ras in dog thyrocytes.

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Pozniak CD, Radinovic S, Yang A, McKeon F, Kaplan DR, Miller FD. An anti-apoptotic role for the p53 family member, p73, during developmental neuron death. Science 2000;289:304-306.

p53 is a tumor suppressor protein. A large number of functions have been attributed to p53, including apoptosis, DNA repair and

maintenance of genetic stability. p73 and p63 are members of the p53 family. p73 and p63 genes have several alternatively spliced mRNA transcripts. Pozniak and collaborators have investigated whether a truncated isoform of p73 (lacking the transactivation domain) plays an antiapoptotic role or not and they have also explored the role p73 and its truncated isoform plays during mouse development.

Expression of p73 variants in the neonatal mouse brain and in the sympathetic superior cervical ganglia was characterized by RT-PCR and analysed by one and two - dimensional Western blot analysis. Full length isoforms and truncated forms (Δ Np73) were expressed in the developing brain and superior cervical ganglion (SCG). Δ Np73 was characterized as the predominant isoform in neonatal sympathetic neurons *in vivo* and in cultured cells.

An adenovirus delivery system was used to maintain expression of Δ Np73 after NGF withdrawal.

This study has demonstrated that adenovirus-infected sympathetic neurons were rescued from cell death; whereas, control adenovirus-infected sympathetic neurons were not. Truncated p73 was also found to prevent cell death during normal development of sympathetic neurons *in vivo*.

In conclusion, Pozniak et al. have demonstrated that Δ Np73, an isoform of p73, inhibits p53 mediated neuronal apoptosis.

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Mainprize KS, Gould SWT, Gilbert JM. Surgical management of polypoid lesions of the gallbladder. Br J Surg 2000; 87: 414-417.

As ultrasound technology improves an increasing number of polypoid lesions of the gallbladder (PIGs) are being detected. They are often incidentally identified during radiographic

evaluation of abdominal pain. The distinction between benign, malignant and potentially malignant lesions is a major diagnostic dilemma and proper management of these lesions remain controversial.

In a recent study performed by Mainprize et al, 38 patients with ultrasonographically detected PLGs were reviewed for patient demography, symptoms, radiological and pathological findings. Thirty-six patients out of 38 were symptomatic of whom 34 had symptoms that could be attributable to the gallbladder. Those 34 symptomatic patients underwent cholecystectomy. There were only 11 who had macroscopic and histopathologically proven PLG in the operative specimen, giving a sensitivity of ultrasonography of 32 per cent. Of these, seven had cholesterol polyps, two had adenomas, one had a carcinoid tumour and one had a tubulo villous adenoma with a focus of invasive adenocarcinoma. One patient had a histopathologically normal gallbladder. The remainder had chronic cholecystitis with or without gallstones. The malignancy rate was therefore 3 per cent (one of 34).

There was no difference in the mean age at presentation of those with chronic cholecystitis, neoplasia or non-neoplastic polyps, although the only adenocarcinoma was diagnosed in the oldest patient (80 years). All of the patients with neoplastic lesions of the gallbladder had solitary polyps greater than 1.0 cm in diameter, whereas those with non-neoplastic PLGs all had multiple lesions less than 1.0 cm in diameter.

PLGs are being found more frequently owing to improvements in ultrasound technology. Their reported frequency varies greatly, from 0.004 to 13.8 per cent of resected gallbladders.

Cholesterol polyps are the commonest form of PLG, responsible for over 60 percent of resected lesions. Benign true tumours and malignant tumours occur with a similar frequency of 5-10 percent. This is a problem as it is very difficult to distinguish benign polyps from malignant or potentially malignant ones on ultrasonography. Ultrasonography has a reported sensitivity of 45 to over 90 percent whereas oral cholecystography has a sensitivity of only 20 percent in most series.

Endoscopic ultrasonography differentiates among polypoid lesions more precisely than ultrasonography (97% vs. 71%). Computed tomography has a reported sensitivity of 60 percent and endoscopic retrograde cholangiopancreatography is of little help.

Factors that increase the chances of a polyp in the gallbladder being malignant include age greater than 50 years, the presence of a single polyp, a polyp greater than 1.0 cm in size, the presence of gallstones, a sessile lesion even if less than 1.0 cm in size, and a rapid change in size on serial ultrasonography.

Taking into account the inaccuracy of ultrasonography in the diagnosis of these lesions, authors have proposed a protocol for the management of ultrasonographically detected PLGs. In this protocol it is suggested that all patients with a PLG should undergo surgery if they are symptomatic, or if the PLG is 1.0 cm or more in diameter. As the sensitivity of ultrasonography is variable, asymptomatic patients are rescanned and the patient is offered a laparoscopic cholecystectomy if a PLG is identified a second time. If the repeat ultrasonographic scan does not show a PLG then a further scan is performed 6 months later and the patient is discharged if no PLG is seen a second time. Patients declining operation are followed with 6-monthly scans and if the lesion increases in size they are once again offered surgery.

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Kraimps JL, Bouin-Pineau MH, Mathonnet M, et al. Multicenter study of thyroid nodules in patients with Graves' disease. Br J Surg 2000;87:1111-1113.

Thyroid nodules are common in patients with Graves' disease. The authors have previously reported that patients with Graves' disease and a thyroid nodule have a very high risk of thyroid cancer. The aim of this study was to review

patients who had surgery for Graves' disease associated with thyroid nodules, and to evaluate the risk of thyroid carcinoma...

Five hundred and fifty seven patients who had thyroidectomy for Graves' disease were studied retrospectively. All patients had undergone surgery because of recurrence of hyperthyroidism after 18 months of medical treatment or because a thyroid nodule was detected and each patient underwent clinical, biochemical, ultrasonographic and scintigraphic evaluation. Surgery consisted of either a subtotal or a total thyroidectomy.

In 140 patients, a thyroid nodule was detected before operation (25.1 percent of population). Thyroid carcinoma was diagnosed histologically in 21 patients, always inside a nodule. The overall incidence of thyroid carcinoma associated with Graves' disease was 3.8 percent, rising to 15.0 percent in patients with Graves' disease and a thyroid nodule. Pathological findings consisted of 20 papillary and 1 follicular carcinoma.

In conclusion this report confirms that one-quarter of patients with Graves' disease have associated thyroid nodules. 15.0 percent of these nodules were malignant. Because of the high incidence of carcinoma in a thyroid nodule, the authors and others recommend surgery when nodules are detected during evaluation for Graves' disease. Total thyroidectomy should be considered because of the aggressive nature of carcinomas in this condition.

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Fon LJ, Spence RAJ. Sportsman's hernia. Br J Surg 2000;87:545-552.

This review article briefly says that keep in mind that there are conditions such as osteitis pubis and musculotendinous injuries other than hernia that make chronic groin pain. Sportsman hernia is a debilitating condition which presents as chronic groin pain. A tear occurs at the external oblique which may result in an occult hernia. The definition, investigation and the treatment of this condition remain unclear. In many cases, clinical signs are lacking despite the patient's symptoms and it is known that there is a high incidence of symptomatic impalpable hernia in patients with obscure groin pain. A medline search was performed from 1962 to 1999 pertaining to chronic groin pain, groin injury and sportsman's hernia. Routine use of CT and MR for assessment of patients with groin pain cannot be justified. They may, however, be employed in difficult cases to help define the anatomical extent of a groin injury. There is no consensus view supporting any particular surgical procedure for sportsman's hernia. Appropriate repair of the posterior wall results in therapeutic benefit in selected cases.

In conclusion the diagnosis of sportsman's hernia is difficult. The condition must be distinguished from the more common osteitis pubis and musculotendinous injuries. When conservative management has failed, surgical intervention is usually, although not always, successful.

MEETINGS

4 - 5 January 2001, London, UK

IBDG January Meeting - Copper Chemistry in Biology and Medicine

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21 - 24 March 2001, Istanbul, Turkey

Heart & Brain

5th International Conference on Stroke & 2nd Conference of The Mediterranean Stroke Society

Contact: Stroke 5 Conference c/o Kenes International - Professional Congress Organizers (PCO) and
International Association Management, P.O.Box 5006, Tel Aviv 61500, Israel
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24 - 30 March 2001, Edinburgh, UK

Society for General Microbiology 148th Ordinary Meeting: New Challenges to Health

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ANSWER TO PHOTO QUIZ

Congenital constriction band syndrome

This is a congenital deformity of the extremities. Incidence is 1:875 births. Two common features of this syndrome are acrosyndactyly and acral absences. As the remnants of web spaces there are rings on the extremities. There is a swelling distal to the ring and the proximal part of it is normal. Neural tube defects, craniofacial abnormalities and abdominal and thoracic wall defects can occur with extremity deformities.

Simple constriction often does not require treatment. Treatment is indicated more on a cosmetic than on a functional basis. For a simple constriction ring, staged excision of the ring and staged Z-plasty of the defect can be done. Rings with large defects can be reconstructed by local flaps. In the case of increasing edema and decreasing circulation, amputation may be considered. Associated congenital abnormalities must be treated.

Author Index for Volume XIII

	Issue, Page		Issue, Page	
Ak, Koray	3: 153		Bilsel, Serpil	2: 115
Aka, Nurettin	4: 219		Binöz, Sedef	4: 196
Akalın, Figen	4: 223		Budak, Erdal	2: 91
Akbulut, U. Günter	2: 75		Cabadak, Hülya	4: 243
Akça, Özlem	1: 30		Canpolat, Cengiz	2: 114
Akdaş, Atif	2: 59		Caymaz, Oğuz	1: 33
Aker, Ömer	4: 205		Civşlek, Ali	3: 153
Aker, Rezzan	3: 127		Çakın, Ayla	3: 131
Akgün, Serdar	3: 153		Çam, Kamil	2: 59
Akın, Levhi	2: 64		Çatav, Zeki	3: 143
Akın, Serap	3: 127		Çavdar, Safiye	2: 115
Akoğlu, Emel	4: 226		Çelebiler, Özhan	3: 176
Aksoy, Serdar	4: 244		Çerikcioğlu, Nilgün	4: 201
Aksu, M. Burak	2: 116		Çobanoğlu, Adnan	4: 233
Aksu, M. Feridun	2: 91		Demiroğlu, Cem'i	3: 137
Aktan, A. Özdemir	1: 22		Demiroğlu, I.C. Cemşid	3: 137
Aktan, Sevinç	1: 19; 3: 148		Dirik, M. Zafer	2: 82
Alkan, Mualla	1: 7		Erdem, Aysun	4: 191
Alnıgeniş, Nergis	3: 169		Erdem, İlknur	4: 191
Alper, Gülay	1: 45		Erdoğan, Tibet	1: 27; 4: 205
Anveriazar, Muhammed	3: 166		Erenus, Mithat	1: 47; 3: 178; 4: 196
Aras, Necdet	4: 205		Erin, Nuray	3: 127
Aslan, Neslihan	3: 127		Erkan, Elif	2: 70
Avşar, Erol	4: 212		Erkan, Özlem	2: 94
Ay, Binnaz	1: 15		Eroğlu, Eren	4: 205
Ayanoğlu, Elif	3: 162;		Erol, Nurdan	3: 156
Ayhan, Çağla	2: 64		Erzik, Can	3: 156
Aytekin, Saide	3: 137		Eşen, Yüksel Mehmet	2: 94
Aytekin, Vedat	3: 137		Eti, Zeynep	1: 15
Bakır, Mustafa	2: 114		Fak, A. Serdar	1: 15, 33
Balkan, Emel	4: 219		Gençosmanoğlu, Rasim	4: 212
Baltacıoğlu, Feyyaz	3: 153		Gezer, Altay	2: 91
Barlan, Işıl	2: 70		Ghandour Salah	2: 64
Başaran, Müjdat	2: 70		Göğüş, F. Yılmaz	1: 15
Başdemir, Demet	2: 70		Göktaş, Paşa	4: 191
Batman, Çağlar	3: 162		Gören, Zafer	3: 127
Bavbek, Tayfun	3: 166		Güleryüz, Meliha	1: 19
Bayramiçli, Mehmet	4: 241		Güllüoğlu, Bahadır	1: 22
Bekiroğlu, Nural	3: 148		Günel, İnce Dilek	1: 19; 3: 148;
Berkman, Kemal	3: 127		Günaydın, Serdar	3: 143
Beşikçi, Resmiye	4: 223		Güney, İlter	3: 131
Bihorac, Azra	4: 226		Gürbüz, Jasna	1: 36, 2: 88
Bilgen, Hülya	4: 201		Gürler, Ayşegül	1: 47; 3: 178; 4: 196

<u>Issue, Page</u>	
Haklar, Goncagül	1: 11
Hamzaoğlu Över, Hülya	4:212
Harmancı, Hande	2:100
İskender, Ece	3:127
Ispir, Turgay	2: 70
Ispir, C. Selim	3:153
İmer, Bahadır	3:176
İnanlı, Selçuk	3:162
Kalaça, Çağrı	2: 82
Kalaça, Sibel	2: 82 ; 3: 131
Kalaycı, Cem	4:212
Kan, Beki	3:131
Kaplancan, Tansel	4:205
Karadağ, Ahmet	2: 64
Karakoç, Birgül	2:109
Karavuş, Ahmet	2: 75
Karavuş, Melda	2: 75
Karcioğlu, Özgür	1: 38
Kaya, Esin	2: 82
Kayaalp, Nimet	2: 94
Kavak, Neşe Zehra	2:109
Kazokoğlu, Haluk	3:166
Keser, İbrahim	1: 7 , 30
Kılıç, Bülent	2: 82
Kılıçaslan, Işın	4:226
Koç, Mehmet	4:226
Köksal, İ. Türker	1: 27
Küçükkaya, Bahire	4:242
Lüleci, Güven	1: 7 , 30
Madazlı, Rıza	2: 91
Maden, Emin	2: 82
Nurichalichi, Kerim	3:148
Oktay, Ahmet	1: 33
Oktay, Şule	3:127
Onat, Filiz	1: 46 ; 3: 127
Onmuş, Hale	2: 75
Orhan, İrfan	1: 27
Orun, Oya	4:242
Ögünç, Güner	2: 98
Özcan, Faruk	1: 27
Özdemir, Cevdet	2:114
Özdoğan, Osman	4:212
Özek, Eren	4:201
Özel, Barut Yıldız	4:191
Özener, İshak Çetin	3:166 ; 4: 226
Özer, Enver	3:162
Özer, Kürşat	3:156
Özgün, Selin	4:241

<u>Issue, Page</u>	
Özgüner, Ahmet	3:156
Öztunç, Funda	4:223
Parmaksızoğlu, Teoman	4:245
Sarı, İbrahim	2: 82
Saybaşılı, Hale	1: 11
Sönmezşık, Gülşen	4:230
Söyletir, Güner	4:201
Süzer, Kaya	3:143
Şad, Orhan	4:212
Şapcı, Tarık	2: 75
Şehitoğlu, Mehmet Ali	3:162
Sirikçi, Önder	3:137
Tarcan, Tufan	3:177
Tefekli, Ahmet	1: 27
Tekeli, Atike	3:153
Tekin, Sabri	2: 98
Tellioğlu, Tahir	3:127
Tezcan, Hakan	1: 33
Tokmakoğlu, Hilmi	3:143
Topçu, Güler	3:137
Topçu, Tuğba	4:243
Toprak, Ahmet	1: 33
Tözün, Nurdan	3:131 ; 4: 212
Tuncer, Neşe	3:148
Tutkun, Alper	3:162
Türkeri, Levent	2: 59
Türkmen, Aysu	3:156
Umuroğlu, Tümay	1: 15
Urgancı, Nafiye	2: 94
Usta, Mustafa	1: 27
Ülger, Nurver	4:201
Üneri, Cüneyt	3:162
Vural Doğan, Sema	2: 94
Vural Tuzcular, Zeynep E.	4:219
Yakut, Cevat	1: 33
Yalçın, A. Şûha	1: 11
Yalın, Aymelek	1: 36 ; 2: 88
Yaman, Hakan	4:230
Yavrucu, Serpil	3:156
Yazıcıoğlu, Eşref	4:219
Yeğen, Ç. Berrak	2: 64
Yılmaz, Berka	4:241
Yılmaz, Yüksel	1: 45
Yimen, Bahadır	4:241
Yorgancıoğlu, Cem	3:143
Yüksel, Mehtap	1: 36 ; 2: 88
Yüksel, Meral	1: 11

Contents of Volume XIII

From the Editor Nurdan Tözün	5
Original Articles	
The results of molecular and cytogenetic analysis in 6 families with fragile – X syndrome in Turkey İbrahim Keser / Güven Lüleci / Mualla Alkan	7
Superoxide radical generation in rat striatal slices: Effects of depolarization and calcium ion deficiency conditions Hale Saybaşıllı / Meral Yüksel / Goncagül Haklar / A. Süha Yalçın	11
The effects of endotracheal intubation and laryngeal mask airway on the risk of myocardial ischemia in cardiac patients Binnaz Ay / Zeynep Eti / A. Serdar Fak / Tümay Umuroğlu / F. Yılmaz Göğüş	15
Trinucleotide repeat length and clinical progression in Huntington's disease Dilek İnce Günal / Meliha Gülerüz / Sevinç Aktan	19
Does the quantity of scientific publications reflect the quality? A rising issue for promotion in developing countries Bahadır M. Güllüoğlu / A. Özdemir Aktan	22
The presence of hydronephrosis in staging bladder cancer: An ominous sign İ. Türker Köksal / İrfan Orhan / Ahmet Tefekli / Mustafa Usta / Tibet Erdoğru / Faruk Özcan	27
Case Reports	
Low IgA associated with short arm deletion of chromosome 18 İbrahim Keser / Güven Lüleci / Özlem Akça	30
Right ventricular myxoma infiltrating the tricuspid valve and obstructing the right ventricular inflow and outflow tracts Hakan Tezcan / Oğuz Caymaz / Ahmet Toprak / Ali Serdar Fak / Cevat Yakut / Ahmet Oktay	33
Thyroid gland with a separate left lobe Aymelek Yalın / Jasna Gürbüz / Mehtap Yüksel	36
Review Articles	
How to consider and manage brain death in an emergency setting Özgür Karcıoğlu	38
Photo Quiz	45
Medicine Elsewhere	46
Meetings	50
Announcement	51
	249

From the Editor Nurdan Tözün	57
Original Articles	
Prediction of insignificant prostate cancer in men with stage T1C disease Kamil Çam / Levent Türkeri / Atif Akdaş	59
The influence of altered thyroid state on gastrointestinal motility in rats Çağla Ayhan / Salah Ghandour / Levhi Akın / Ahmet Karadağ / Berrak Ç. Yeğen	64
Blood lead levels of children in Istanbul who work at high risk jobs Elif Erkan / Demet Başdemir / Işıl Barlan / Turgay İspir / Müjdat Başaran	70
Impression cytology (IC) in clinical practice Tarık Şapçı / Ahmet Karavuş / Hale Onmuş / Melda Karavuş / Uğur Günter Akbulut	75
What do our patients know about diabetes mellitus? Sibel Kalaça / Çağrı Kalaça / Esin Kaya / Bülent Kılıç / İbrahim Sarı Emin Maden / M. Zafer Dirik	82
Case Reports	
Lumbar arteries with uncommon patterns of origin Jasna Gürbüz / Mehtap Yüksel / Aymelek Yalın	88
A giant leiomyoma originating from the rudimentary uterine buds in Rokitansky-Kuster-Hauser syndrome M. Feridun Aksu / Rıza Madazlı / Altay Gezer / Erdal Budak	91
Hepatocellular childhood carcinoma with a presentation of two cases Nafiye Urgancı / Özlem Erkan / Sema Doğan Vural Mehmet Yüksel Eşen / Nimet Kayaalp	94
Surgical skills	
Laparoscopic second - look under local anaesthesia after bowel resection Güner Öğünç / Sabri Tekin	98
Review Articles	
The global polio eradication program: Current situation in strategies and achievements in the world and in Turkey Hande Harmancı	100
Antenatal corticosteroids for fetal maturation Birgül Karakoç / Neşe Zehra Kavak	109
Photo Quiz	114
Medicine Elsewhere	115
Meetings	118

From the Editor Nurdan Tözün	125
Original Articles	
Plasma concentration-time profile of a single dose of enteric-coated omeprazole in male and female healthy volunteers Ece İskender / Neslihan Aslan / M. Zafer Gören / Tahir Telliöđlu / Serap Akın Nuray Erin / Rezzan Aker / Filiz Onat / Kemal Berkman / Şule Oktay	127
Students' perceptions on medical education at Marmara University School of Medicine Sibel Kalaça / Beki Kan / Ayla Çakın / İlder Güney / Nurdan Tözün	131
Homocysteine is not an indicator of restenosis risk after percutaneous transluminal coronary angioplasty Önder Sirikci / Vedat Aytakin / Güler Topçu / Saide Aytakin I.C. Cemsid Demirođlu / Cem'i Demirođlu	137
Free flow capacity of internal thoracic artery grafts after sodium nitroprusside injection to the pedicle Cem Yorgancıođlu / Hilmi Tokmakođlu / Serdar Günaydın / Zeki ÇataV / Kaya Süzer	143
The clinical profile of nonmotor fluctuations in Parkinson's disease patients Dilek İnce Günal / Kerim Nurichalichi / Neşe Tuncer / Nural Bekirođlu / Sevinç Aktan	148
Case Reports	
Axillocephalic arteriovenous graft: A new alternative for hemodialysis Atike Tekeli / Serdar Akgün / C. Selim İsbir / Ali Civelek Koray Ak / Feyyaz Baltacıođlu	153
Bardet - Biedl syndrome Nurdan Erol / Aysu Türkmen / Ahmet Özgüner / Serpil Yavrucu / Can Erzik / Kürşat Özer	156
Fibrous dysplasia of the temporal bone Selçuk İnanlı / Enver Özer / Elif Ayanođlu / Alper Tutkun / Çađlar Batman Cüneyd Üneri / M. Ali Şehitođlu	162
Renal transplantation and cytomegalovirus retinitis Tayfun Bavbek / Muhammed Anveriazar / Haluk Kazokođlu / Çetin Özener	166
Review Articles	
Clinical significance of antiperinuclear factor and antikeratin antibody for rheumatoid arthritis M. Nergis Alnigeniş	169
Photo Quiz	176
Medicine Elsewhere	177
Meetings	182

From the Editor Nurdan Tözün	189
Original Articles	
Central venous catheter related infections in haemodialysis patients İlknur Erdem / Aysun Erdem / Paşa Göktaş / Yıldız Barut-Özel	191
Effect of adding alendronate to hormone replacement therapy on bone mineral density in established postmenopausal osteoporosis Mithat Erenus / Ayşegül Gürlü / Sedef Binöz	196
Neonatal candida infections in an intensive care unit: a three year experience Hülya Bilgen / Eren Özek / Nurver Ülger / Nilgün Çerikcioğlu / Güner Söyletir	201
Evaluation of acute flank pain with non-contrast spiral CT and its predictive role on clinical outcome Tibet Erdoğru / Tansel Kaplancan / Necdet Aras / Ömer Aker / Eren Eroğlu	205
Percutaneous endoscopic gastrostomy: results of 50 cases Rasim Gençosmanoğlu / Orhan Şad / Erol Avşar / Hülya Över Hamzaoğlu Osman Özdoğan / Cem Kalaycı / Nurdan Tözün	212
The risk factors in postmenopausal osteoporosis Nurettin Aka / Emel Balkan / E. Zeynep Tuzcular Vural / Eşref Yazıcıoğlu	219
Case Reports	
Cor triatriatum associated with Ebstein malformation of atretic mitral valve and double outlet right ventricle Funda Öztunç / Figen Akalın / Resmiye Beşikçi	223
5-Aminosalicylic acid associated chronic tubulointerstitial nephritis in a patient with Crohn's disease Mehmet Koç / İshak Çetin Özener / Azra Bihorac / Işın Kılıçaslan / Emel Akoğlu	226
Transient neonatal pustular melanosis Gülşen Sönmezşık / Hakan Yaman	230
Perspectives in cardiac transplantation: Operative techniques and early postoperative care in cardiac transplantation Adnan Çobanoğlu	233
Photo Quiz	241
Medicine Elsewhere	242
Meetings	246
Author index for volume XIII	247
Contents of volume XIII	249



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Contents

From the Editor	
<i>Nurdan Tözün</i>	189
Central venous catheter related infections in haemodialysis patients	
<i>İlknur Erdem / Aysun Erdem / Paşa Göktaş / Yıldız Barut-Özel</i>	191
Effect of adding alendronate to hormone replacement therapy on bone mineral density in established postmenopausal osteoporosis	
<i>Mithat Erenus / Ayşegül Gürler / Sedef Binöz</i>	196
Neonatal candida infections in an intensive care unit: a three year experience	
<i>Hülya Bilgen / Eren Özek / Nurver Ülger / Nilgün Çerikcioğlu / Güner Söyletir</i>	201
Evaluation of acute flank pain with non-contrast spiral CT and its predictive role on clinical outcome	
<i>Tibet Erdoğan / Tansel Kaplancan / Necdet Aras / Ömer Aker / Eren Eroğlu</i>	205
Percutaneous endoscopic gastrostomy: results of 50 cases	
<i>Rasim Gençosmanoğlu / Orhan Şad / Erol Avşar / Hülya Över Hamzaoğlu / Osman Özdoğan / Cem Kalaycı / Nurdan Tözün</i>	212
The risk factors in postmenopausal osteoporosis	
<i>Nurettin Aka / Emel Balkan / E. Zeynep Tuzcular Vural / Eşref Yazıcıoğlu</i>	219
Cor triatriatum associated with Ebstein malformation of atretic mitral valve and double outlet right ventricle	
<i>Funda Öztunç / Figen Akalın / Resmiye Beşikçi</i>	223
5-Aminosalicylic acid associated chronic tubulointerstitial nephritis in a patient with Crohn's disease	
<i>Mehmet Koç / İshak Çetin Özener / Azra Bihorac / Işın Kılıçaslan / Emel Akoğlu</i>	226
Transient neonatal pustular melanosis	
<i>Gülşen Sönmezışık / Hakan Yaman</i>	230
Perspectives in cardiac transplantation: Operative techniques and early postoperative care in cardiac transplantation	
<i>Adnan Çobanoğlu</i>	233
Photo Quiz	241
Medicine Elsewhere	242
Meetings	246
Author index for volume XIII	247
Contents of volume XIII	249